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THE OCCURRENCE OF THE RARER DEMYELINATING DISEASES (NEUROMYELITIS OPTICA, SCHILDER'S ENCEPHALITIS ET CETERA) IN SOUTH AUSTRALIA.

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WHILE it is our impression that disseminated sclerosis is much less prevalent in South Australia than in London or in Melbourne, cases of the rarer demyelinating diseases occur with some frequency. Thus de Crespigny and Woollard[1] published the first record of a case of human Schilder's disease in Australia. Holmes à Court and Latham[2] and Penington[3] have described further cases occurring in this country. Hurst and McLennan[4] also observed a case in a monkey long resident in the Adelaide Zoological Gardens. The following three specimens came to the pathological laboratories of the Institute of Medical and Veterinary Science within approximately a year; one is a case of *neuromyelitis optica*, one is a case of Schilder's *encephalitis periaxialis diffusa*, and one is difficult to classify, though it is probably related to the latter condition. Cox[5] mentioned having seen a case of *neuromyelitis optica* in Melbourne, though, as far as we know, the present is the first fully documented in Australia.

Case I.

Mrs. E.M. was forty-six years of age and had given birth to four children. In July, 1935, her vision became misty, but improved after a few days. She then developed what she called influenza, which left her somewhat weak. At the end of August sight again began to fail, and by

the beginning of September mere perception of light remained. Examined now by Dr. Michael Schneider, the right eye was blind, without perception of light. The left eye recognised moving objects held one foot before it; the pupil reacted sluggishly to light and immediately dilated. The fundi showed no abnormality. At this time no other abnormal neurological signs were noted, and the cerebro-spinal fluid was normal. The patient developed what appeared to be acute cholecystitis, which cleared up under medical treatment, and later acute tonsillitis.

In October vision began to improve, and by October 26, seven weeks after the first examination, was $^6/_9$ in the right eye and $^6/_{36}$ partly, in the left eye. The fields showed very slight peripheral loss with a central colour scotoma. A month later the temporal sides of the disks were paler than before, and the patient complained a little of glare, for which she needed tinted glasses.

In April, 1936, vision was unaltered and general health excellent. In July, vision failed completely in both eyes and the optic nerve heads were redder than formerly. After three weeks in hospital recovery had taken place to such an extent that vision in the left eye was $^6/_9$ and in the right eye $^6/_{12}$, and the patient went home (August 7). Three weeks later she returned completely blind, with no reaction to light in the right eye and only an occasional reaction in the left. She complained now of girdle pains.

After temporary improvement during the following month the vision of the right eye was $^6/_{36}$ and of the left eye $^6/_{12}$. The right optic disk became white and the vessels diminished in calibre; at the end of 1936 this eye was completely and finally blind from optic atrophy. The left eye retained some vision until the terminal stages of the illness.

In September, 1936, retention of urine developed, and later in the month the patient complained of pains in the lumbar region, spreading into the buttocks, thighs and legs.

During October and November power and sensation in the lower limbs gradually diminished; all forms of sensation were equally impaired. At the same time muscular power was lost—at first dorsiflexion of the foot, then plantar flexion and finally all movements of feet, ankles, knees and hips. The last movements to disappear were flexion of the metatarso-phalangeal joints of the great toes. The deep reflexes were over-active at first, but in the last two or three weeks of life they disappeared. Plantar reflexes were extensor. With the onset of more flaccid paralysis, the early retention of urine gave place to constant dripping incontinence.

Pain was a persistent and most distressing symptom. The pains were of two kinds, sharp and shooting, and dull and aching; spreading from the back into the lower limbs, they were sufficiently severe to make the patient, a stoical woman, cry out. These persisted even after all movement and sensation in the lower limbs had been lost. The upper limit of anæsthesia was at the level of the sixth dorsal segment on both sides. The upper limbs and thoracic muscles were unaffected. During the last month vision was lost completely and the lower part of the trunk and the lower limbs were without sensation or motion. For the last two weeks muttering delirium was constant, and death occurred on February 3, 1937, from exhaustion hastened by urinary sepsis.

Examined on October 6, 1936, the cerebro-spinal fluid failed to react to the Wassermann test and contained 70 milligrammes *per centum* of protein, with a slight excess of globulin. Cells were normal in number. The colloidal gold curve was 1112221000.

On November 7, 1936, the fluid, still clear and colourless, contained 100 milligrammes *per centum* of protein and a definite excess of globulin. The cells, nearly all lymphocytes, numbered 20 per cubic millimetre. The colloidal gold curve was 1233432000.

Pathological Examination.

The brain, brain-stem, upper cervical and mid-dorsal cord, in a state of moderately good preservation, were available for section and were stained by the usual neuropathological methods. The dorsal cord was definitely shrunken.

Generalized Changes.—All regions, whether otherwise affected or not, showed widespread but irregular perivascular cuffing with lymphocytes; arteries and veins alike were involved. In general the white matter was more affected than the grey, though many vessels in both escaped. Cuffing was not very intense; although two or three layers of cells might be present in the Virchow-Robin space, the whole circumference of the vessel was not always covered. In the meninges, particularly over the base of the brain, similar cuffing occurred, and there were stretches of lymphocytic infiltration in many places. In a few areas at the base slight microglial increase was noted in the cortical zone immediately underlying the meninges. Mucoid degeneration of the oligodendroglia was generalized in the white matter. In all parts of the cortex and basal ganglia the nerve cells were singularly well preserved.

Around very occasional infiltrated veins in the white matter slight microgliosis was evident. A narrow zone of demyelination, associated with much microglial increase, surrounded a single small vessel situated immediately below the cortex in one occipital lobe, very much as in post-vaccinal and other similar forms of encephalitis.

In the reticular formation of the pons and medulla a few microglial nodules were present, while in the floor of the fourth ventricle there occurred a marked increase of rod cells around some vessels and also diffusely in the grey matter. Rare nerve cells appeared to be undergoing degeneration, but the vast majority were very well preserved.

Localized Changes.—The optic nerves, optic chiasma and optic tracts were almost completely demyelinated. In preparations stained by Weil's method the last could be seen entering the region of the external geniculate ganglia as very pale bands in an otherwise normally myelinated territory. Degeneration was not detected beyond the ganglia. Further forward in the chiasma and optic nerves demyelination was still more complete and axis cylinders were wholly absent. Perivascular cuffing was no more intense than elsewhere in the brain, though in places some lymphocytes overflowed into the nervous tissue. The microglia was considerably increased, as rod cells in the nerve generally and as foci of granular corpuscles around some vessels and in the connective tissue sheath of the nerve. Fibre-forming glial cells, sometimes with large bodies, and abundant fibrils were present, and newly deposited collagen fibrils were also conspicuous. It was impossible to obtain any indication of the earlier distribution of the lesions.

In the first cervical segment of the spinal cord ascending degeneration was very evident in the postero-internal columns and was present also in the antero-lateral columns, where, however, it was perhaps hardly commensurate with the intensity of degeneration in lower segments. In the second cervical segment, immediately ventral to the tip of one posterior horn, there existed in addition a small focus of destruction of myelin and axis cylinders; this area resembled those in the dorsal cord (see below). In both segments nerve cells were normal.

In the stretch of dorsal cord examined no part was normal, and the most affected levels showed almost complete disorganization of structure across most of the transverse section. At these levels the pathological process apparently had its origin centrally, and, even when the white matter was involved, was furthest advanced there; the whole cord was shrunken, the grey matter more so than the white. A highly cellular area of destruction covering most of the grey matter sent finger-like processes towards the surface, to join with independent foci in the white matter. Irregular areas of recognizable white matter remained as islands in the areas of maximum change and near the surface of the cord. Thus the lesions were only very roughly symmetrical in the two halves of the cord, and they were wholly unsystematized.

As seen peripherally in parts of the white matter, the early changes apparently took the form of destruction of

myelin sheaths and axis cylinders, with subsequent overgrowth of microglia, slight at first, but rapidly becoming intense. They were often, but not always, perivascular in distribution, and by coalescence a large area of complete demyelination and of destruction of axis cylinders surrounding several large vessels often resulted. In these areas rod cells and granular corpuscles laden with neutral fat and doubly refractile lipoid were numerous. A few lymphocytes flowed over from infiltrated perivascular spaces and some large fibre-forming glial cells were present. In some affected areas of the grey matter a few more or less degenerate nerve cells persisted; in uninvolved parts the nerve cells were normal. A minority of arterioles had thickened hyaline walls, but most were not narrowed and the main spinal arteries were normal; there was thus no evidence of vascular occlusion as an ætiological agent. In many places in the affected areas considerable new formation of small and greatly dilated vessels was associated with deposition of abundant collagen fibrils in the nervous tissue proper. The meninges of the affected segments were often free from change, or at most showed a scanty lymphocytic infiltrate in the pial meshes.

Case II.

Miss M.L., aged twenty years, whose occupation was the performance of home duties, was treated for squint from the age of six months and lost the sight of the right eye at three years. Otherwise she had no history of illness, and as a child showed intelligence and musical ability.

About three months before admission to hospital her ankles began to turn over and her vision to fail. Two months later she began to experience headache on the side of the head and behind the eyes, and thereafter became increasingly apathetic. The left leg became weak, tremulous and painful. She vomited once.

On admission to hospital on January 20, 1937, the patient was stuporose, with the head tilted to the left. Exophoria of the right eye and bilateral ptosis were present. Movements of the eyes were full laterally, with coarse nystagmus more pronounced when she looked to the left. Movements in the vertical plane were slight and produced coarse vertical nystagmus. The left pupil was larger than the right; the left reacted to light, the right only consensually. The pupils did not respond to attempts at accommodation. The left side of the face was atonic and slightly paretic. The tongue, deeply furrowed and brown, was protruded in the middle line; the palate was raised symmetrically and the palatal reflex was strong. When the arms were outstretched the left fell slightly and became hyperpronated; the left wrist was atonic. The abdominal muscles were board-like in rigidity and the abdominal reflexes diminished on the left. Tonus in the legs was normal. All tendon reflexes were exaggerated, especially on the left side; ankle clonus was more marked on the left. The plantar responses were equivocal. Examination of sensory functions was unsatisfactory, but demonstrated no gross disturbance.

Dr. A. L. Tostevin reported the presence of old disseminated retino-chorioiditis in the right eye and found nothing to explain loss of vision in the left eye. The visual fields could not be mapped out, owing to lack of cooperation on the part of the patient.

The cerebro-spinal fluid, under normal pressure and clear and colourless, contained 140 milligrammes *per centum* protein and definite excess of globulin; it had a normal cell count and the colloidal gold curve was 0012221000. Except for a fall in the protein content to about half the above figure, these findings remained substantially the same throughout the patient's illness. The Wassermann test applied to both blood and fluid yielded no reaction.

Further developments in the clinical condition were as follows.

On February 4, 1937, the patient had a "trembling" attack, repeated next day, appeared very drowsy and vomited.

From February 8 to 15, 1937, there was tonic reflex of the fingers of the left hand, the movements of which became clumsy and ill-directed. Objects could be recognized by touch (but not always named), but not by vision. There was tonic extension of the left arm and leg, with sustained knee and ankle clonus. The patient rubbed her nose frequently. The right leg moved freely in response to stimuli, the left very little. Abdominal reflexes disappeared.

From February 22 to March 8, 1937, the patient became still more drowsy and was very restless; she rolled the head continually from side to side, bringing the right arm and leg up and down while the limbs of the other side were immobile. She made no response to questions and moaned constantly. She kept the mouth firmly closed and had to be fed forcibly. An intravenous injection of 0·1 gramme of "Neo-silver-salvarsan" was given on February 25, 1937.

From March 23, 1937, improvement set in, particularly of the mental condition, and by April 5, 1937, exercises and massage were begun. No nystagmus persisted and eye movements were full.

From April 15 to 22, 1937, the patient could now see and grasp a hand. Abdominal reflexes had reappeared on the right side. The sense of touch and pinprick was less acute in the left leg, vibration sense was present and warmth could be distinguished from cold.

Improvement was maintained for several months in spite of the fact that primary optic atrophy could be detected in the left eye (July 1, 1937). Three weekly doses of 0·2 gramme of "Neo-silver-salvarsan" were given intravenously during August. On September 13, 1937, the patient could draw crudely a triangle, a square and a tennis racket. Asked to draw a bucket, she thrice drew a tennis racket. She recognized a match only after taking it out of the box, when she struck the match and blew it out. She named a pen after much hesitation, and allowed a watch to be named a button. On October 6, 1937, she was comparatively well, though unable to sit up; she took an interest in her surroundings, listened to the radio and recognized colours. From October 20 to 24, 1937, she menstruated for the first time since the onset of her illness. By November 15, 1937, she could sit out of bed, but had little power in the legs. There was diminution of all forms of sensation in the left leg and an extensor plantar response was present on this side. A further intravenous injection of 0·15 gramme of "Neo-silver-salvarsan" was administered on November 22, 1937.

Drowsiness and spasticity of the left leg now increased again, the temperature rose, signs of pneumonia appeared, and the patient died with a temperature of 40·5° C. (105° F.) on December 8, 1937. With the exception of this terminal pyrexia there had been only occasional rises to 37·2° or 37·8° C. (99° or 100° F.) during the preceding January and February, and once in April.

Throughout the illness the patient had been incontinent of urine and fæces. Constipation was severe, usually yielding only to glycerine and oil enemata given every third day, except during periods of a week or more, when saline draughts or cascara proved effective.

Pathological Examination.

Much of the brain and some of the cord were available for examination. The remainder of the cord was kept for inoculation into rabbits, mice, guinea-pigs and monkeys (*Macaca mulatta* and *Macaca irus*); the results were uniformly negative. The macroscopic appearances in the brain were typical of Schilder's disease. Numerous "plaques" were present also in the brain-stem and cord.

Microscopic Examination.—Large, continuous and roughly symmetrical areas of complete demyelination spread forward from the ventral half of the white matter of the occipital lobes; here in many places even the arcuate fibres were not preserved, but the cortex was not appreciably involved. The edge of the demyelinated area against the normal white matter was as sharp as that of a plaque in disseminated sclerosis. Further forward (on the right side) the area tended to hug the wall of the ventricle where this was lined by white matter, and then at the level of the basal nuclei split into two main tongues. The larger dorsal, covering the medial and

upper part of the cerebral white matter from the vertical cortex to the lateral ventricle and head of the caudate nucleus, extended through the ventral part of the *corpus callosum* to the opposite hemisphere and anteriorly to within about an inch of the frontal pole. The arcuate fibres were wholly absent, without extension of the demyelinating lesion into the cerebral cortex. A smaller ventral tongue invaded the white matter of the frontal lobe below the caudate nucleus.

In these areas a few axones persisted, some showing varicosities or terminal swellings. In some parts of the lesion, presumably the older, large fibre-forming and often multinucleated glial cells were abundant, as were also large glial nuclei with no very distinct cell body but with many fibrils. "Globoid" cells (Collier and Greenfield[20]) were few and far between. Various forms of microglial cell containing products of myelin degeneration were numerous; the latter were crystalline and anisotropic to a considerable extent. Some of the perivascular spaces were filled with granular corpuscles containing fat, others with lymphocytes, while in still others both types of cell were present. In a few areas many new vessels had developed, and even occasional fine collagen fibrils radiating into the nervous tissues. In contrast to the state of affairs in the preceding case, perivascular infiltration was not seen in regions distant from the demyelinated area.

In a few areas obviously more recently affected, for example, some of the processes of white matter extending into the gyri from the *centrum semiovale*, fibre-forming glial cells were replaced by large *gemästete* glia cells, and the perivascular spaces contained few *Gitterzellen*; here destruction of myelin was often incomplete.

In a few otherwise normal parts of the cortex overlying the demyelinated zone, irregular deposits of calcium and iron salts apparently represented calcified nerve cells and also cells much more irregular and highly branched (? microglial cells); small granules and calcified globules also lay free in the tissues. Hitherto we have not observed this phenomenon in Schilder's disease, though it is not infrequent in the cortex of idiot children dying within a short time of birth, and in the regions surrounding vascular lesions of the brain.

In the brain stem many areas of complete demyelination, varying from 0·2 to 1·0 centimetre in length and up to 0·3 centimetre in breadth, were sharply demarcated from their surroundings. They were not especially localized in the neighbourhood of the fourth ventricle. Fibre-forming glial cells without large bodies were very numerous, and fibrils were much more abundant than in the cerebrum. No globoid cells were present.

Much the same appearances obtained in several areas in the cerebellar white matter, where new-formed vessels together with glial cells and fibrils arranged in piloid manner were the chief elements in the parts of the plaques adjoining the fourth ventricle. At the margins in contact with the normal white matter myelin degeneration was more recent, granular corpuscles abundant and glial fibrils almost absent. In other parts tiny foci of early demyelination appeared to have resulted from the coalescence of a number of small perivascular areas; here the products of breakdown of the myelin still stained with hæmatoxylin.

In transverse sections of the spinal cord from several levels areas of demyelination were evident. Since most of the cord was kept in glycerine for experimental purposes, we could not decide whether or not the areas at various levels were separate or connected. They were certainly not of equal duration, since some showed only early and partial demyelination and others complete demyelination with some new formation of small vessels. While at many levels the demyelination was confined to the white matter, particularly of the posterior columns, at others the grey matter was more affected, although nerve cells in an infiltrated area were uniformly well preserved. The cord lesions were not more symmetrical than those in the preceding case of *neuromyelitis optica*. Glial cells of any variety were not nearly so conspicuous as in the cerebrum of the present patient. Perivascular cuffing with lymphocytes was intense around some vessels.

Case III.

Mrs. G.H., aged thirty-eight years, had had one child. About two months before her admission to hospital she noticed an abnormal tendency to fatigue; she yawned frequently and was disinclined to effort. Three weeks before her admission the right side of the mouth was "raised" as she talked. Ten days later the left hand became weak, followed by the left arm and the legs, with a tendency to trip over objects. There was nausea but no vomiting, and no headache or disturbance of any sensation.

On her admission to hospital on October 5, 1937, signs of a left flaccid hemiparesis were more marked in the upper than in the lower limb, and were accompanied by no sensory loss. The left abdominal reflexes were absent, all tendon reflexes were increased, and the plantar reflexes were flexor. On the right side there was ptosis. A slight defect in articulation was considered due to the facial weakness. The visual fields and fundi were normal.

The cerebro-spinal fluid on October 8, 1937, was clear and colourless and contained three lymphocytes per cubic millimetre. A slight excess of globulin accompanied a protein content of 30 milligrammes *per centum*. The colloidal gold curve was 1233200000. The Wassermann test yielded no reaction.

The report on the encephalogram (October 20, 1937) stated that no air had entered the right lateral ventricle, and that there was atrophy of the right frontal lobe along the vertex.

Between the date of admission and October 26, 1937, the patient developed numbness, paræsthesia and spasticity in the left arm. The left leg was weaker than before and was still flaccid, but an extensor plantar reflex with tendency to ankle and patellar clonus was evident. The right ptosis became more marked. Yawning and grinding of the teeth were frequent. Some psychical disturbance—somnolence, untidiness and unreasonable requests—was now manifest.

On November 6, 1937, an exploratory craniotomy was performed by Mr. Leonard Lindon, following which the woman developed a purulent meningitis and died eight days later.

Pathological Examination.

The brain, but not the cord, was available for section.

Macroscopic Changes.—Apart from a generalized purulent meningitis, the brain, when sectioned after hardening in formol-saline solution, showed the following changes. The outlines of the right *globus pallidus* were obscured, and with the putamen this nucleus appeared less pink and more translucent than normal. More anteriorly the yellowish-grey area affected most of the internal capsule and spread into the white matter above and laterally to the head of the caudate nucleus, flanking ultimately the lateral ventricle. A similar area involved the external capsule and claustrum. These areas were distinctly softer than normal.

On the left side similar changes were less extensive and confined to the ventral part of the internal capsule and the anterior part of the *globus pallidus*. The left side of the brain appeared larger than the right, and anteriorly on this side the ventricle was represented by a narrow slit, while on the other side it was a normal cavity. Since this appearance conflicted with the encephalographic findings, it is not clear how far it represented an artefact or an effect of the operation.

Microscopic Changes.—Microscopic examination confirmed in essentials the distribution of changes noted macroscopically. Except for some cuffing of the superficial parts of cortical vessels with polymorphonuclear cells, due to an extension inwards of the meningeal infiltrate, lesions were limited to the deeper regions mentioned and to one or two adjacent foci. Their general character, the overgrowth of fibrous glia and the stage which myelin breakdown had reached, no less than the clinical history of the case, proved that they antedated considerably the purulent meningitis which developed during the last eight days of life.

The vessels of affected regions, but not those elsewhere, were often heavily cuffed with lymphocytes and occasional plasma cells. In some places granular corpuscles containing neutral fat were also present in the perivascular spaces. The nervous tissues showed complete demyelination accompanied by overgrowth of microglial cells containing lipoid material, much of it crystalline and anisotropic. With these mingled occasional lymphocytes and plasma cells, and many large, sometimes multinucleated, glial cells with abundant fibrils. No "globoid" cells were noted. In a few areas the large glial cells extended into adjacent white matter well beyond the limits of the demyelinated areas. Though by the standard established by Greenfield and King[7] damage to axones could be classed as severe, a number of these structures persisted in all but the most damaged areas and showed varicosities and *boules de rétraction*. In the most affected regions of the *globus pallidus* a number of nerve cells remained quite well preserved, with normal nuclei and only partial disappearance of the Nissl substance.

As might be expected, the extent of these changes was rather greater than suggested by the macroscopic appearances. In places there also existed secondary tract degeneration (as in the fibre bundles of the left putamen and in the fornix of this side). The degree of demyelination in the right internal capsule was wholly disproportionate to the partial paralysis obtaining during life, and was in itself evidence of a primary attack on the myelin, with much less injury to the nerve fibres themselves. Further evidence of this was seen in a few tiny outlying foci of demyelination, or merely of ballooning of myelin sheaths, in the mesencephalon *et cetera*. These were accompanied by scarcely any loss of axis cylinders, were clearly perivenular in distribution, and with their cuffs of lymphocytes in the perivascular spaces and the accumulation of microglial phagocytes in the extraadventitial tissues they bore striking resemblance to the lesions of post-vaccinal encephalitis. It appeared not improbable that the major bilateral and very roughly symmetrical lesions in the basal ganglia represented a confluence of such perivascular lesions.

Calcification of the vessels of the anterior half of the *globus pallidus* and numerous calcified mulberry bodies here completed the pathological picture.

Comment.

Recent years have seen growing interest in the diseases of the central nervous system characterized by a primary demyelination, the classical example of which is, of course, disseminated sclerosis. This disease appears to be distinctly uncommon in South Australia; but if our recent experience is a true criterion, the rarer diseases of the group occur with a frequency out of all proportion to the incidence of disseminated sclerosis. Without information as to the full ætiology of these demyelinating maladies, scientific endeavour has been restricted to clinical differentiation of various syndromes, and to classification according to the type and distribution of the pathological changes. In their description of familial Schilder's disease, Meyer and Tennent[8] draw attention to the insufficiency of histological classification, while in his comprehensive review of the group Ferraro[9] suggests that artificial distinctions have thus been created by the association of the names of authors with what may be only rather peculiar variants of one disease.

The three cases described in this paper exhibit the following characteristics:

1. Bilateral, more or less symmetrical lesions, usually showing complete demyelination and often, when not too advanced, only partial destruction of axones or nerve cells.

2. The usual reactive changes on the part of the microglia with considerable proliferation of large fibre-forming glial cells.

3. Infiltration of the perivascular spaces with inflammatory cells. All cases showed a tendency to some lymphocytic exudation in the demyelinated zones.

4. Evidence, derived from occasional early lesions away from the main areas of damage, of a perivascular distribution of the demyelination, a distribution best seen in post-vaccinal and similar forms of encephalomyelitis, but also described by one or other author in almost all the demyelinating diseases. The observation suggests perhaps that extension and coalescence of such zones may contribute materially to the formation of the main areas (compare Stewart, Greenfield and Blandy,[10] and others).

In addition, the first and second cases presented in differing degree an overgrowth of mesenchyme in the form of new vessels and of deposition of collagen in the nervous tissues.

The first case is a typical example of the condition known as *neuromyelitis optica* (and by numerous synonyms). When describing such a case in 1927, Beck[11] cited seventy possible cases in the literature. Later, Balser[12] collected records of four cases and stated that between 1927 and the time when he wrote his paper (1936) about forty fresh cases had been reported. To us it appears not improbable that an essentially similar pathological condition occurs even more frequently than these figures indicate. Except for the more acute course (three months) the case described by Környey[13] as belonging to the group of "central leucomyelitides" closely resembles the one under consideration in the nature of the lesions in the cord, though in the absence of involvement of the visual apparatus the striking clinical syndrome of *neuromyelitis optica* was not reproduced. Identification of an ætiological agent would make it possible to determine how significant is the present classification of demyelinating diseases upon a clinical or clinicopathological basis. It is interesting to note that in our first case, as in those of Beck and Balser, perivascular cuffing with lymphocytes was general throughout the nervous system, whereas in Cases II and III it was localized fairly rigidly in the demyelinated areas.

Case II is fairly typical of Schilder's *encephalitis periaxialis diffusa*, and was in fact diagnosed as such during life. It is well known that remissions may occur in the clinical course of the malady, as in this case.[6][14][15] On the pathological side it is equally well recognized that the brain stem, cord *et cetera* may show areas very similar to those in disseminated sclerosis.[10][14] It is interesting perhaps in the present case that, judging by the relative absence of lipoid and the dense nature of the glial sclerosis, the areas in the cerebellar white matter appeared to be at least as old as or even to antedate those in the cerebral hemispheres. The arcuate fibres are not always spared by the cerebral

lesion,[14][15] and in our case they were particularly severely affected.

As has not infrequently happened with Schilder's encephalitis,[14] in Case III the symptoms pointed to the existence of a cerebral tumour of which the exploratory craniotomy and the subsequent autopsy revealed no trace. Instead, the lesions found were those of a primary demyelinating disease; the reasons for this conclusion are set forth in the histological description. Without a more comprehensive viewpoint than is commonly adopted, classification of this case would be difficult. For although it is well recognized that apart from main lesions in the white matter of the cerebrum in Schilder's disease, areas of the cerebral cortex, brain-stem, cord and optic nerves may be affected, no case of Schilder's disease exactly like the present has been recorded. Histologically, except for the absence of "globoid cells", the lesions were not dissimilar to those of Schilder's encephalitis, yet no changes were present in the occipital lobes, and in the region of the basal ganglia the grey matter was at least as severely affected as the white, especially on the left side. The *corpus callosum* was uninvolved. Whether in the interest of unity and simplification this case be classed as one of atypical Schilder's disease or whether it be raised to the dignity of a new entity is a matter of taste. Our own feeling is that in the present state of our knowledge, and on the basis of possibly unimportant differences in the site of a lesion and its finer histopathology, it is unwise to add further to the numerous variants of the better known demyelinating diseases. We are inclined therefore to regard this case as essentially one of atypical Schilder's disease.

Summary.

Three cases of the rarer primarily demyelinating diseases are described; they occurred in South Australia within the space of one year. One is a case of *neuromyelitis optica* and one of Schilder's *encephalitis periaxialis diffusa*. The third does not correspond exactly with any in the literature, but we are inclined to regard it as an atypical case of Schilder's disease.

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PERFORATION OF THE STOMACH AND DUODENUM DUE TO SIMPLE ULCER AND CARCINOMA.

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Introduction.

I am presenting the records of a personal series of operations for perforation of the stomach and duodenum in the hope that an analysis of the deaths and the results may contribute something to the treatment of this disaster. In the main my conclusions only confirm many well-known facts about the management and prognosis; but I think that such confirmation and repetition are desirable from time to time, especially when they derive from personal experience as against mass statistics.

These 83 patients came under my care between the years 1925 and 1938. Twenty-five died, either directly as a result of the perforation or from later complications or from other disease. I shall consider separately the three cases of perforated carcinoma, which were all fatal. This leaves us with 80 cases of simple ulcer of the stomach or duodenum, with 22 deaths, a mortality rate of 27·5%. Of these 80 patients, 63 had duodenal ulcer and 17 gastric ulcer. The 63 patients with duodenal ulcer were all males (I have never seen a female with a proven duodenal ulcer). Twelve of these 63 died, the mortality rate thus being 19%.

I have divided the duodenal ulcer cases into two groups:

Group A (Cases I to XXXIII): The patients were under my care at Saint James's Hospital, London, and comprise my first 33 patients.

Group B (Cases XXXIV to LXIII): The patients were Sydney residents and form my next 30 patients.

Of the 33 patients in Group *A*, nine died (a mortality rate of 27·3%). Of the 30 patients in Group *B*, three died (a mortality rate of 10%). The great difference in the death rate in these two groups is discussed later.

Of the 17 patients with gastric ulcer, 10 died (a mortality rate of 58·8%). Ten of the 17 were males, and six (60%) died. Seven of the 17 were females, and four (57·1%) died.

Note that the mortality rate for perforated gastric ulcer (58·8%) is much higher than for perforated duodenal ulcer (19%).

Further analysis shows why the grand mortality for perforated peptic ulcer (gastric and duodenal combined) was as high as 27·5%.

(*a*) One death may be classed as inevitable, namely, that of a man who survived perforation of a duodenal ulcer, to die several months later with neoplasm of the liver (Case VI).

(*b*) Five males and one female were virtually moribund on admission to hospital and might justifiably have been refused operation. Perforation had occurred 3 days, 25 hours, 3 days, 26 hours, 24 hours and 21 hours, respectively, before operation. Of the males, the first two had gastric ulcer (Cases XIII and XIV) and the remaining three duodenal ulcer (Cases I, II and III). The female had a gastric ulcer (Case XIX).

(*c*) A female with gastric ulcer died on the seventh day after operation, from profuse hæmatemesis (Case XX).

(*d*) A preventible but fortuitous death resulted from strangulation of small gut in a suprapubic stab wound, from which the tube had been removed three days before. The patient was a male with a gastric ulcer (Case XVIII).

If the patient cited in paragraph (*a*), who did not die of perforation, is disregarded, the mortality rate is reduced from 27·5% to 26·5%. On subtraction also of the six moribund patients of paragraph (*b*), the mortality rate is 20·5%. On subtraction of the death from hæmatemesis mentioned in paragraph (*c*), the mortality rate is 19·4%. On subtraction also of the death from intestinal strangulationed mentioned in paragraph (*d*), the mortality rate is 18·3%.

The last figure, 18·3%, represents the mortality rate in my series of operations on the "ordinary risk" type of patient with perforated peptic ulcer.

Amended Mortality Rate.

Now let us apply these subtractions to the duodenal and gastric ulcer series separately.

Mortality from Perforated Duodenal Ulcer.

The gross mortality rate from perforation of duodenal ulcer is 19%. On subtraction of the patient of paragraph (*a*), the mortality rate is 17·7%. On subtraction also of the three moribund patients of paragraph (*b*) the mortality rate is 13·5%.

The patient of paragraph (*a*) belonged to Group *A*. On subtraction of this case the mortality rate of Group *A* drops from 27·3% to 25%. Two of the patients of paragraph (*b*) belonged to Group A, and when these also are subtracted the mortality rate for Group *A* becomes 20%.

One patient of paragraph (*b*) belonged to Group *B*, and on subtraction of this case the mortality rate for Group *B* drops from 10% to 6·9%.

Summary of Records of Mortality from Perforated Duodenal Ulcer.

Gross mortality for perforated duodenal ulcer ..	19·0%
Amended mortality for perforated duodenal ulcer	13·5%
Gross mortality for Group *A* (Cases I to XXXIII)	27·3%
Amended mortality for Group *A* (Cases I to XXXIII)	20·0%
Gross mortality for Group *B* (Cases XXXIV to LXIII)	10·0%
Amended mortality for Group *B* (Cases XXXIV to LXIII)	6·9%

Mortality from Perforated Gastric Ulcer.

The gross mortality rate from perforation of gastric ulcer is 58·8%; the amended mortality rate is 41·6%; thus on subtraction of the three moribund patients of paragraph (*b*) the mortality rate drops to 50%; on subtraction also of the death from hæmatemesis mentioned in paragraph (*c*), the mortality rate is 46·1%; on subtraction of the death from intestinal strangulation noted in paragraph (*d*), the mortality rate is 41·6%.

Records of Fatal Cases.

Let us briefly review the records of the fatal cases, namely, twelve of duodenal ulcer and ten of gastric ulcer, and see what can be learnt from them. Can we do anything to reduce the death rate of perforation?

Perforated Duodenal Ulcer.

Three patients, mentioned in paragraph (*b*), died of shock and sepsis, which were present before operation (Cases I, II and III below). One died suddenly and unexpectedly from probable pulmonary embolism (Case IV). One died from neoplasm of the liver (Case V). One died from bronchopneumonia (Case VI). Four died from general or spreading peritonitis, not present before operation, in contrast with Cases I, II and III (Cases VII, VIII, IX and X). Two died of subphrenic abscess (Cases XI and XII).

In Case III the site of the perforation was not determined; in all the others it was on the anterior wall of the first part of the duodenum.

Case I.—A male, aged forty years, was admitted to hospital on the third day after perforation. The usual signs of abdominal catastrophe were present. The temperature was 38·3° C. (101° F.), the pulse rate was 130, and respiration rate 36 per minute. The patient was cyanosed and sweating. Dulness to percussion and bronchial breath sounds were detected at the bases of both lungs, ascribed to lobar collapse.

Provisional Diagnosis: Perforated ulcer or acute pancreatitis.

Operation: A local anæsthetic and a minimal amount of open ether were used. The perforation was closed. Local peritonitis only was observed. Suprapubic drainage was employed.

Result: The patient died three days after operation.

Autopsy: Subhepatic abscess and moderate peritonitis were observed. The small gut was inflamed and distended. Closure of the perforation was intact. A small non-perforating ulcer was observed on the posterior wall of the duodenum. The lower lobe of each lung was collapsed.

Case II.—The patient was a male, aged about sixty years, who gave a vague history. Operation was performed twenty-six hours after perforation. The patient was a fat man, very ill, with a dry tongue and the usual signs of perforation.

Operation: Much free gas, bile and duodenal fluid were found. The perforation was closed; suprapubic drainage was employed.

Result: The patient died eight hours after operation.

Autopsy: The perforation was soundly closed. There was a collection of bile in the subhepatic area, and fat necrosis of the omentum had occurred.

In both these cases epigastric drainage would have been more efficacious than suprapubic drainage. Note that there was no gross general peritonitis in either of these cases, in contrast with Cases VII, VIII, IX and X.

Case III.—A male, aged forty-five years, was operated on twenty-four hours after perforation. The patient was desperately ill. He was dehydrated and suffering from shock.

Operation: Under local anæsthesia, epigastric and suprapubic drainage was performed. No attempt was made to locate the ulcer. Much free fluid was observed in the belly.

Result: The patient died eight hours after the operation. There was no autopsy.

Case IV.—The patient was a male, aged seventy years. Operation was performed within twelve hours of perforation. The perforation was closed. No drainage was employed. The patient suffered from post-operative bronchitis, but was apparently doing well when he died suddenly on the twelfth day.

Autopsy (limited to the abdomen): The perforation was in an acute ulcer of the duodenum and was well closed. There were two smaller acute ulcers in the pyloric area of the stomach. Some lymph was found on the upper surface of the liver, but no abscess or collection of fluid anywhere. The general peritoneal cavity was clean. The cause of death was uncertain, but the mode of death was suggestive of pulmonary embolism.

Case V.[1]—The patient was a male, aged thirty-seven years, who was operated on within twelve hours of perforation. The perforation was closed. The ulcer was not noticeably indurated. Nodules of growth on the upper surface of the liver were observed. The patient was readmitted two months later with an enormous nodular liver; his general condition was poor.

Autopsy: Autopsy was performed nineteen weeks after the operation. The liver was big and largely replaced by neoplasm, which to microscopic examination was sarcoma or immature carcinoma. No primary lesion was found in the abdomen. A simple chronic ulcer of the duodenum was present.

Case VI.—The patient, a male, aged forty-five years, was operated on about twelve hours after perforation. Closure of the ulcer was effected; posterior gastro-enterostomy was performed. No drainage was employed. Three days after the operation he developed a cough productive of purulent sputum; his temperature rose and respiration rate increased. Dulness to percussion was detected at the base of the left lung. There were no symptoms or signs of abdominal disorder. He died eight days after the operation.

Autopsy: Closure of the ulcer and the anastomosis were satisfactory. There was no peritonitis; no fluid was found in the abdomen. Purulent bronchopneumonia of the bases of both lungs, worse on the left side, was found.

Case VII.—The patient was a male, aged twenty-five years. Operation was performed sixteen hours after perforation. Bile-stained fluid was observed escaping through the hole. No drainage was employed. Twelve days later, when apparently convalescing normally, the patient developed symptoms and signs of another perforation. At operation several hours later, lymph and adhesions were found around the duodenum; but there was neither free gas nor fluid and no perforation was to be seen. He died the next day.

Autopsy: General peritonitis and much fluid were present, in contrast with findings at the operation the day before. The original perforation, identified by suture material, was apparently soundly closed. There was a small hole on the upper border of the duodenum; but it was doubtful whether this was due to acute ulceration or to injury during separation of the stomach and duodenum from the liver. The other abdominal organs were intact.

Case VIII.—A male, aged forty-eight years, was operated on about seventeen hours after perforation, which had occurred during a paroxysm of vomiting. His condition was poor and the operation was done under local and splanchnic anæsthesia without any general anæsthesia. No drainage was established. He died twenty-seven hours later. Prior to his death he became cyanotic, his pulse became rapid, and his blood pressure fell.

Autopsy: Black fluid was seen all over the peritoneum. The perforation, which was in a large chronic ulcer, seemed to be well closed; but the stitches separated easily during removal of the stomach and duodenum, and there was not yet any sign of natural healing or "sealing off" of the perforation. This was probably due in part to *post mortem* digestion.

Case IX.—A male, aged forty-two years, was operated on eight hours after perforation. No drainage was established. He died on the second day.

Autopsy: General peritonitis was present. The sutures in the ulcer were intact and the hole was closed; but, as in Case VIII, there was little evidence of "sealing off". There was another ulcer on the posterior wall of the duodenum.

Case X.—The patient was a male, aged forty-five years. He had had an operation for perforation of a duodenal ulcer eight months previously. The second operation was performed seven hours after perforation. Much fluid and some bismuth were seen in the abdominal cavity. Drainage was not used. He died the next day.

Autopsy: General peritonitis and subphrenic abscess were observed. The ulcer was adherent to the gall-bladder. The perforation seemed to have been well closed; but leakage occurred during removal of the duodenum. Note the early formation of subphrenic abscess.

Case XI.—The patient was a male, aged sixty years. Operation was performed twenty-four hours after perforation. He made good progress for nine or ten days, and then symptoms and signs of right subphrenic abscess developed, but no pus was found on exploration. He died twenty-six days after operation.

Autopsy: A right anterior intraperitoneal subphrenic abscess was present. Closure of the chronic duodenal ulcer was secure; but there were few signs of healing.

(Note: This patient should not have been allowed to die without drainage of the abscess.)

Case XII.—A male, aged sixty-seven years, was operated on twenty-four hours after perforation. He was very ill, and operation was done under local infiltration and gas and oxygen anæsthesia. Fat necrosis of the omentum was noted. No drainage was employed. He became mentally confused soon after the operation and was febrile; there were no obvious abdominal symptoms or signs. He gradually failed and died on the eleventh day.

Autopsy: The ulcer was found firmly closed by the sutures; but when they were removed the hole gaped and there was no sign of natural healing. (Compare Cases VIII, IX and XI.) The ulcer was moderately acute, and there was another acute ulcer on the posterior wall. No fat necrosis was evident. (Compare operation findings.) A large right intraperitoneal subphrenic abscess was present.

Summary of the Fatal Cases of Duodenal Perforation.

A summary of the twelve fatal cases of perforated duodenal ulcer is given in Table I.

Gastric Ulcer Group.

Males Who Died from Perforated Gastric Ulcer.

Six males in the gastric ulcer group died. Two, mentioned in paragraph (*b*), died of sepsis and shock, which were present before operation (Cases XIII and XIV). Two died of peritonitis (Cases XV and XVI). One died of bronchopneumonia and pelvic peritonitis (Case XVII). One, mentioned in paragraph (*d*), died of intestinal strangulation through a stab drainage wound (Case XVIII).

Case XIII.—A male, aged sixty years, was operated on three days after perforation. He was apparently moribund. The temperature was 36·1° C. (97° F.) and the pulse rate 120 per minute. The belly was full of turbid fluid and lymph. The stomach was adherent to the liver; it was not disturbed, in view of the patient's desperate condition. Simple drainage was employed. The patient died several hours later.

Autopsy: There was a perforation in a chronic ulcer on the lesser curvature of the stomach. Collapse of the lower lobe of the left lung had occurred.

[1] I reported this case in The Medical Journal of Australia (August 4, 1928) in a paper entitled "Double Lesions".

TABLE I.
Summary of the Twelve Fatal Cases of Perforated Duodenal Ulcer.

Case Number.	Sex.	Age.	Interval Between Perforation and Operation.	Drainage.	Interval Between Operation and Death.	Cause of Death.
I	M.	40	3 days.	Yes.	3 days.	Preoperative sepsis and shock.
II	M.	60 (about)	26 hours.	Yes.	8 hours.	Preoperative sepsis and shock.
III	M.	45	24 hours.	Yes. (alone).	8 hours.	Preoperative sepsis and shock.
IV	M.	70	12 hours. (about)	No.	12 days.	Pulmonary embolism.
V	M.	37	12 hours. (about)	No.	19 weeks.	Neoplasm of liver.
VI	M.	45	12 hours.	No.	8 days.	Bronchopneumonia.
VII	M.	25	16 hours.	No.	13 days. (after first operation)	General peritonitis.
VIII	M.	48	17 hours.	No.	27 hours.	General peritonitis.
IX	M.	42	8 hours.	No.	2 days.	General peritonitis.
X	M.	45	7 hours.	No.	1 day.	General peritonitis and subphrenic abscess.
XI	M.	60	24 hours.	No.	26 days.	Subphrenic abscess.
XII	M.	67	24 hours.	No.	11 days.	Subphrenic abscess.

CASE XIV.—The patient was a male, aged fifty-six years, who was operated on twenty-five hours after perforation. His general condition was bad. Free gas and stomach contents and two separate localized collections of turbid fluid were observed in the subhepatic and subphrenic areas. A perforation in a chronic prepyloric ulcer was closed. Three drainage tubes, one subhepatic, one subphrenic and one suprapubic, were inserted. He died three hours later. No autopsy was performed.

CASE XV.—The patient was a male, aged fifty-four years, who was operated on within twelve hours of perforation. An ulcer was found high up on the lesser curvature of the stomach. The perforation was closed with difficulty. Suprapubic drainage was employed. The patient died on the fifth day.

Autopsy: General peritonitis and much free fluid were noted. Fat necrosis of the omentum over the site of the ulcer had occurred. The ulcer was the size of a florin. There was little evidence of healing, and the hole was insecurely closed. There was a smaller ulcer on the lesser curvature, near the pylorus.

CASE XVI.—A male, aged fifty-two years, was operated on six hours after perforation. Hæmatemesis had occurred four days before. Much gas and brownish-yellow fluid were found in the belly. As much fluid as possible was removed with a sucker and saline solution was poured in. No drainage was employed. The stomach was very distended, and as the patient vomited brown fluid during the operation, I washed out the stomach at the conclusion. Next day his condition was satisfactory; but on the second day he collapsed, with sudden abdominal pain, and again presented the clinical picture of a perforated viscus. I reopened the wound without anæsthesia and brown fluid gushed out. The original perforation was found securely closed and the stitches were intact. Fluid continued to well up; but I could not locate the source. As the patient was failing, I packed the omentum over the front of the stomach and duodenum and fixed it with several stitches and inserted two drainage tubes, one at the site of the original perforation and the other into the general peritoneal cavity at the lower end of the wound. The patient died twelve hours later, in spite of the intravenous administration of fluids and a blood transfusion.

Autopsy: There was a chronic ulcer in the pyloric antrum near the lesser curvature. Leakage had apparently been controlled; but when the stitches were removed the perforation looked larger than it did at the first operation. Was this due to trauma, such as from the needle punctures or from stitches that had cut in, or was it due to extension of ulceration? The ulcer was indurated only around the distal three-quarters of its circumference; proximally it did not appear to be chronic. There was localized peritonitis in the right hypochondrium, right iliac fossa and the pelvis.

CASE XVII.—The patient was a male, aged thirty-seven years. Operation was performed ten hours after perforation. Gas and much fluid were observed in the belly. There was a perforation of the anterior wall of the stomach through a large chronic prepyloric ulcer, which was adherent posteriorly. The perforation was closed. Fluid was sucked out of the belly and saline solution poured in. No drainage was employed. The patient died on the fourth day. He was cyanosed and had a mucus rattle in the throat. Crepitations could be heard at the bases of both lungs.

Autopsy (limited to the abdomen): There was a little turbid fluid in the belly. A loop of small gut covered with lymph was adherent to the bottom of the pelvis. There was no peritonitis above the pelvic brim, and the rest of the small gut looked normal; but all the large gut was distended with gas. Suturing of the perforation seemed sound and there was no leakage. The ulcer was just proximal to the pylorus, fixed to the pancreas and extending around the lesser curvature to the anterior wall, where perforation had occurred. The lungs were not examined; but clinically the cause of death was bilateral bronchopneumonia. The operation was done under local anæsthesia supplemented by nitrous oxide and oxygen anæsthesia and preceded by an injection of morphine, hyoscine and atropine.

CASE XVIII.—The patient was a male, aged fifty-four years. Operation was performed about seven hours after the perforation of a chronic prepyloric ulcer on the lesser curvature. I closed the hole without difficulty, and then, as the belly was flooded with gastric contents and fluid, I made a suprapubic stab wound for a half-inch drainage tube; this was removed thirty-six hours after operation. The patient made good progress until the fifth day, when 22·5 centimetres (nine inches) of small gut were extruded through the stab wound, which had apparently healed. The gut was gangrenous by the time I was summoned, and the patient died two days after resection, that is, a week after the first operation. (I reported this case in *The Journal of the College of Surgeons of Australasia*, Volume II, Number 3, March, 1930: "A Rare Cause of Intestinal Strangulation".)

Autopsy: General peritonitis and ileus.

Females Who Died from Perforated Gastric Ulcer.

Four females in the gastric ulcer group died. One, mentioned in paragraph (*b*), died of sepsis and shock, present at the time of operation (Case XIX). One, mentioned in paragraph (*c*), died of post-operative hæmatemesis (Case XX). One died from shock and the results of preceding hæmatemesis (Case XXI). One died from pneumonia (Case XXII).

CASE XIX.—A female, aged forty-four years, was subjected to operation twenty-one hours after perforation. Her condition was poor. There were much gas and fluid in the belly and deposits of lymph on the liver and

stomach. Perforation on the anterior wall of a chronic saddle ulcer of the stomach was closed. No drainage was provided. She died thirty-six hours after operation.

Autopsy: The perforation was soundly closed. A little fluid lay between the liver and stomach. The ulcer was adherent to the pancreas.

Case XX.—The patient was a female, aged fifty years, who was operated on about twelve hours after the perforation of a chronic ulcer about the centre of the lesser curvature of the stomach. There was a small amount of free gas and fluid. No drainage was employed. Hæmatemesis occurred on the fifth and sixth days, and she died on the seventh day.

Autopsy: The perforation was well closed. There was no peritonitis. The ulcer was adherent to the pancreas and had eroded an artery of medium size.

Case XXI.—A female, aged fifty-eight years, was operated on eight hours after perforation. She had been admitted to hospital twenty-four days before on account of hæmatemesis and melena, and was still in hospital when the perforation occurred; it was the anterior wall of a saddle ulcer adherent to the pancreas. No drainage was used. She died nine hours after operation. No autopsy was performed.

Case XXII.—A female, aged forty-eight years, was operated on five hours after perforation. Free fluid and stomach contents were observed in the belly. The ulcer was high up on the lesser curvature; there was little induration around it. Fluid was sucked out and saline solution was poured in. No drainage was employed. She made good progress for a fortnight. On the fifteenth day there were fever, cough and sputum, and dulness to percussion at the base of the left lung, with tubular breath sounds and crepitations. She died eighteen days after operation.

Autopsy: The perforation was well closed. There were two ulcers, fairly acute, in the stomach. The rest of the abdomen was clear. The lower lobe of the left lung was solid and airless, and some of the bronchi were filled with purulent fluid.

Summary of the Fatal Cases of Perforated Gastric Ulcer.

A summary of the fatal cases of perforated gastric ulcer is given in Table II. For comparison, Table III gives a record of the seven successful cases.

Drainage and Mortality.

I have already divided the series of 63 perforated duodenal ulcers into two groups: *A*, Cases I to XXXIII (Saint James's Hospital, London); *B*, Cases XXXIV to LXIII (Sydney). Drainage was employed in five of the 33 cases in Group *A*, namely, two of the nine fatal cases (Cases I and II), three of the twenty-four successful cases. These three were amongst my earliest cases; I am sure now that drainage could have been safely omitted in two of them. In the third perforation had occurred thirty-six hours before operation and there was much free fluid all over the belly. I should still insert a drainage tube under similar circumstances, but in the epigastrium instead of in the hypogastrium. (See below.)

Table III.

Summary of the Essential Features of the Seven Successful Cases of Operation for Perforated Gastric Ulcer.

Sex.	Age.	Interval Between Perforation and Operation.	Drainage.	Remarks.
		Hours.		
M.	54	6	No.	
M.	35	4	No.	
M.	20	16	No.	Lymph plastered over tiny perforation, with little leakage.
M.	55	9	No.	
F.	56	12	No.	
F.	44	9	No.	
F.	30	4	No.	Successful partial gastrectomy two years later.

In Group *B* (30 cases) there were three deaths. Drainage was employed in one of the fatal cases (Case III, drainage being the only operative treatment), but in none of the 27 successful cases.

Note that in all three of the fatal cases in which drainage was used, the patients were classified as moribund on admission (Cases I, II and III), with preoperative infection as a result of the long perforation-operation interval. Could drainage have saved any of the patients who died without drainage? I think that the four patients (Cases VII to X) who died of general peritonitis would not have survived even with drainage; but the two who died from subphrenic abscess (Cases XI and XII) might have survived if I had used immediate epigastric or subphrenic drainage instead of, or as well as, suprapubic drainage.

Let us now consider the seventeen cases of perforated gastric ulcer as a whole. Of the ten fatal cases, drainage was employed in five; of the seven successful cases drainage was employed in none. Subdividing the patients according to sex: ten

Table II.

Summary of the Ten Fatal Cases of Perforated Gastric Ulcer.

Case Number.	Sex.	Age.	Interval between Perforation and Operation.	Drainage.	Interval between Operation and Death.	Cause of Death.
XIII	M.	60	3 days.	Yes. (Alone.)	Several hours.	Preoperative sepsis and shock.
XIV	M.	56	25 hours.	Yes.	3 hours.	Preoperative sepsis and shock.
XV	M.	54	12 hours. (about)	Yes.	5 days.	General peritonitis.
XVI	M.	52	6 hours.	No. (first operation) Yes. (second operation).	3 days.	Peritonitis and shock.
XVII	M.	37	10 hours.	No.	4 days.	Bronchopneumonia.
XVIII	M.	54	7 hours.	Yes.	7 days.	Intestinal strangulation.
XIX	F.	44	21 hours.	No.	36 hours.	Preoperative sepsis and shock.
XX	F.	50	12 hours.	No.	7 days.	Hæmatemesis.
XXI	F.	58	8 hours.	No.	9 hours.	Preceding hæmatemesis and shock.
XXII	F.	48	5 hours.	No.	18 days.	Pneumonia.

were males, of whom six died; five of these six were treated by drainage (one at second operation only—Case XVI). Seven were females, of whom four died; none was treated by drainage. Two of the males who died after drainage were moribund on admission (Cases XIII and XIV). One of the females who died without drainage was moribund on admission.

Drainage did not save any life in this gastric ulcer group; for in none of the seven successful cases was drainage employed, nor do I think that any of the patients who died and who had been treated without drainage could have been saved by drainage, with the possible exception of the patient in Case XVI. It is just questionable that he might have survived without a second operation if drainage had been used for several days after the first operation.

One patient (Case XVIII) died directly as the result of drainage. (See Table II.)

Summary of Records of Perforated Gastric and Duodenal Ulcer.

I now think that drainage should be used where perforation is of long standing (say twelve hours or more), especially if there is much fluid free in the belly. The site of drainage should be the epigastric or subphrenic area rather than, or as well as, the pelvis. In most cases I think that drainage is unnecessary or even harmful. On the contrary, I think that as much fluid as possible should be removed from the belly with a sucker and that several pints of saline solution should be poured in; some escapes, taking with it some of the residual fluid that the sucker misses, and the rest dilutes any that remains and provides the patient with a useful source of physiological fluid. The fear of spreading infection is a bogy. I carried out this procedure in most of my successful cases and in every one of the last twenty-five.

It is clear that the factor of drainage or non-drainage does not account for the great difference in mortality rate between the two groups of the duodenal ulcer series given above. What, then, is the explanation of it?

The first possibilities that come to mind are: (*a*) improved operative technique in the later cases; (*b*) Australian patients stand perforation and operation better than English patients, perhaps because of differences in climate or living conditions; (*c*) longer perforation-operation interval in the cases of Group *A*. We can dismiss this last supposition at once, for examination of my records shows no appreciable difference.

Both the first two propositions seem to be contradicted by the fact that in nine cases in Group *A* I performed gastro-enterostomy immediately after closing the perforation. One patient died (Case VI), the mortality rate thus being 11%. (I performed gastro-enterostomy on the first patient only of Group *B*, and he made a good recovery; since then I have not done it at the time of operating for perforation.) This suggests that my operative technique at that time was satisfactory and that the English patients could not only stand perforation and closure well, but also the additional operation of gastro-enterostomy, even allowing that I selected the patients for it very carefully. I still cannot satisfactorily account for the different mortality rates of Groups *A* and *B*. The figures are too small to be of any statistical value.

This brings us to the question of the advisability and justification of adding gastro-enterostomy to the operation of simple closure of the perforation. As mentioned above, I have done this once only in the thirty cases of Group *B*. This is because I have felt that the main business of operation for perforation is to save the patient's life and because gastro-enterostomy at this time may not only add to the risk of operation, but may not be necessary or desirable for the particular patient, whom we probably have not seen and studied before the perforation. Yet in this small series of ten cases in which I performed gastro-enterostomy at the time of operation for the perforation, one patient only, the first of the series, died—a mortality rate of 10% (see Table IV). If the late results of the survivors, whom I am trying to trace, are good, I should consider performing gastro-enterostomy after closing the perforation, in the following circumstances: (i) if the patient's condition is so good that the extra procedure is not likely to add to his risk; (ii) if also the ulcer is chronic and there is a history of dyspepsia that has not improved with medical treatment; (iii) if there is any evidence of pyloric stenosis, either as a direct complication of the ulcer or as a result of the suturing to close the perforation. Table VI gives the death rate in relation to the perforation-operation interval.

Perforation of Carcinoma of the Stomach.

Perforation of gastric carcinoma is well known, but rare compared with perforation of peptic ulcer. Dr. Cyril Corlette, consulting surgeon to Sydney Hospital, tells me he has never seen such a case. I append brief histories of the three cases of perforation of a carcinoma already referred to at the beginning of this paper.

Case XXIII.—A female, aged fifty-four years, was operated on about nine hours after perforation. The patient was very ill, and closure was carried out under local anæsthesia supplemented by open ether. The perforation was in the centre of a hard mass on the anterior wall of the stomach, near the greater curvature. Saline solution was put into the belly, which was closed without drainage. Four months later the patient agreed to exploratory cœliotomy. Metastases were found in the liver, omentum and parietal peritoneum, none of which were observed at the first operation. She died three months later.

She had suffered from attacks of epigastric pain or distress for ten years, worse in the last two years. The pain used to come three to four hours after food, and especially after the evening meal. She would sometimes be awakened at night by pain and frequently obtained relief by eating. The pain was usually preceded by a feeling of fullness and was usually followed by belching and vomiting. The site of the pain was epigastric, sometimes passing through to the back. After a vomiting attack she used to feel pain in the left shoulder. There had been but little loss of weight. She had been treated in a medical ward with the diagnosis of duodenal ulcer, and had been discharged only about three weeks before the

TABLE IV.

Summary of Results of Gastroenterostomy in Addition to Closure of Perforation of Duodenal Ulcer: Ten Cases.

Sex.	Age.	Perforation-Operation Interval.	Result.	Remarks.
M.	45	12 hours.	Died on eighth day of broncho-pneumonia. (Case VI.)	
M.	41	10 hours.	Recovered.	Six months later: "well."
M.	22	5½ hours.	Recovered.	Five months later: "recent attack of vomiting."
M.	33	12 hours (about)	Recovered.	Good immediate recovery.
M.	50	7½ hours.	Recovered.	Four months later: "no indigestion."
M.	21	5½ hours.	Recovered.	Five months later: "well."
M.	50	8 hours.	Recovered.	Four months later: "occasional digestive upset."
M.	43	4 hours.	Recovered.	Good immediate recovery.
M.	44	4 hours.	Recovered.	Three months later: "well and at work."
M.	48	5 hours.	Recovered.	Good immediate recovery.

TABLE V.

Table of Ages of all Patients with Perforated Peptic Ulcer.

Decade.	Duodenal Ulcer. (All Patients were Males.)		Gastric Ulcer.	
	Successful.	Fatal.	Successful.	Fatal.
0 to 9	——	——	——	——
10 to 19	1, aged 18.	——	——	——
20 to 29	8, aged 21, 25, 22, 21, 24, 20, 23, 26, respectively.	1, aged 25.	1, aged 20, a male.	——
30 to 39	10, aged 32, 30, 30, 33, 31, 36, 33, 30, 34, 35, respectively.	1, aged 37.	2, aged 30 (female), 35 (male).	1, aged 37 (male).
40 to 49	15, aged 44, 41, 41, 41, 43, 43, 41, 44, 48, 40, 40, 42, 46, 41, 47, respectively.	6, aged 40, 45, 48, 42, 45, 45, respectively.	1, aged 44 (female).	2, aged 48 (female), 44 (female).
50 to 59	12, aged 53, 54, 52, 50, 50, 50, 54, 50, 52, 56, 50, 50, respectively.	——	3, aged 56 (female), 54 (male), 55 (male).	6, aged 58 (female), 50 (female), 54 (male), 54 (male), 56 (male), 52 (male).
60 to 69	2, both aged 60.	3, aged about 60, 60, 67, respectively.	——	1, aged 60 (male).
70 to 80	3, aged 72, 70, 78, respectively.	1, aged 70.		

TABLE VI.

Perforation-Operation Interval.	Duodenal Ulcer.		Gastric Ulcer.	
	Fatal Cases.	Successful Cases.	Fatal Cases.	Successful Cases.
Up to 8 hours ..	2	31 (6·1% mortality)	3	3 (50·0% mortality)
8 to 12 hours ..	3[1]	16 (15·7% mortality)	4	3 (57·1% mortality)
12 to 16 hours ..	1	1 (50·0% mortality)	0	1 (0% mortality)
16 to 24 hours ..	2	0 (100% mortality)	1	0 (100% mortality)
Over 24 hours ..	4	3[2] (57·1% mortality)	2	0 (100% mortality)
Totals ..	12	51 (19·0% mortality)	10	7 (58·8% mortality).

[1] One of these patients (Case V) survived his perforation and his operation should be classed as successful in the reckoning of the mortality of perforation *per se*, so that the figure for this eight to twelve hours group would read: Fatal, 2; successful, 17; mortality, 10·5%.

[2] Note that three patients survived perforation of duodenal ulcer of over twenty-four hours' standing. In all three the perforation was tiny and in two of them the perforation was completely covered with lymph. A similar condition was found in the patient who survived perforation of a gastric ulcer of sixteen hours' standing. These cases show that the size of the perforation may be as important in prognosis as the perforation-operation interval.

perforation. Gastric analysis thirteen days before perforation showed hypochlorhydria, and the radiologist's report on an X ray examination eight weeks before perforation read:

> Stomach low and dilated. Gastric hypomobility—six-hour residue. Duodenal ileus.

Gastric analysis about four months after perforation showed achlorhydria, and X ray examination at this time revealed "a large penetrating ulcer extending to lesser curvature, suggestive of malignancy; stomach empties slowly".

Case XXIV.—A female, aged sixty-two years, was subjected to operation four hours after perforation of a prepyloric ulcer of the lesser curvature. No drainage was used. At the time I thought that I was dealing with a large chronic simple ulcer. She promptly recovered, but within a fortnight was complaining bitterly of post-prandial pain. I advised partial gastrectomy, which I carried out a month after the perforation. Microscopic examination showed adenocarcinoma of the stomach and neighbouring glands. She died about a year later from recurrence.

She had suffered from attacks of epigastric pain for eighteen months; these had become worse in the last fortnight. There had been few remissions. She had suffered a moderate loss of weight.

Case XXV.—The patient was a male, aged forty-one years. Operation was performed several hours after perforation, which occurred three or four hours after a diagnostic barium meal. Closure of a perforation in a carcinoma on the anterior wall of the stomach near the lesser curvature was performed. There was a metastatic nodule near the lower edge of the liver, and the neighbouring lymph nodes were large and hard. Drainage was employed. The patient died two days after operation.

For five months before his admission to hospital he had suffered from attacks of epigastric pain, vomiting and loss of weight. A report of an X ray examination made on the day of perforation was: "Organic lesion of the pyloric antrum causing obstruction."

In these three cases the clinical picture of the perforation was indistinguishable from perforation of a simple ulcer. The male patient (see Case XXV) died directly from the perforation; the two females (see Cases XXIII and XXIV) survived perforation, to die of recurrence and extension of the growth.

AN INVESTIGATION OF THE VISSCHER-BOWMAN TEST FOR PREGNANCY.[1]

By VERA I. KRIEGER, D.Sc.,
From the Biochemistry Department, University of Melbourne, and the Women's Hospital, Melbourne.

A SIMPLE chemical test for pregnancy has been described by Visscher and Bowman[1] and the reaction in the test is stated to depend on the oxidation of the anterior pituitary hormone excreted in the urine of pregnant women. The details of the test are as follows:

To one cubic centimetre of urine are added one drop of 1% hydrogen peroxide, five drops of 1% phenyl hydrazine hydrochloride solution, five drops of 5% methyl cyanide, and five drops of concentrated hydrochloric acid. The mixture is heated in a boiling water bath for twenty-five minutes. The result of the test is positive if a reddish brown flocculent precipitate appears. If the precipitate is powdery or absent and the solution is straw coloured the result is negative.

Visscher and Bowman claimed 93% accuracy in 317 proved pregnancies.

Menken[2] reported favourably on the test, but his series was very small.

Dolff[3] obtained positive results in 96% of tests on 54 pregnant women. In extrauterine pregnancies and miscarriages the test was correct in 82% of cases. Dolff states that if the result of the test is negative, pregnancy is absent. He found false positive readings in highly concentrated urine containing reducing katabolic substances and indicates that the test would be more valuable if these could be removed. He regards the test as superior to the Aschheim-Zondek in being simpler, cheaper and requiring less time.

Dodds[4] reported 90% positive results in the testing of 100 pregnant women and 88% accuracy among the non-pregnant. Because of the high percentage of error in the non-pregnant group this writer regards the test as of little real value.

Sheehan[5] reports the following results: males (one to twelve years), 68% positive; males (twelve to eighty years), 62% positive; females (one to twelve years), 80% positive; non-pregnant females (twelve to eighty-one years), 61% positive; females giving a negative response to the Aschheim-Zondek test, 73% positive; females giving a positive response to the Aschheim-Zondek test, 77% positive.

Schenck and Tran[6] reported 16·9% incorrect results in 112 cases, the error being 38·8% among non-pregnant patients.

On investigation of 513 patients Frech[7] found 20 false positive and 20 false negative readings, and he regards the test as less reliable, although cheaper, quicker and simpler than the Aschheim-Zondek test.

Results of the Visscher-Bowman Test Applied to a Series of Pregnant and Non-Pregnant Patients.

Urine from 276 pregnant and non-pregnant patients was submitted to the Visscher-Bowman test and the results are recorded in Table I.

The test was found exceedingly difficult to interpret since it was frequently difficult to distinguish "powdery" and "flocculent" precipitates in such small volumes of liquid. Sheehan[5] found the same difficulty in interpreting the results of his tests and took into account the colour of the urine as well as the precipitate. In the present series, because of the well-known effect of concentrated hydrochloric acid on urinary pigments producing deep reddish and brown colours, the colour and character of the precipitate only were used as the criteria for the determination of the reaction. The results obtained may be divided into the following types: (i) clear solution; (ii) hazy solution; (iii) slight, fine, dark brown precipitate; (iv) heavy, fine, dark brown precipitate; (v) slight brown gelatinous

[1] In a subsequent article V. Krieger will describe the results of an investigation into the Patterson test for the chemical diagnosis of pregnancy.

TABLE I.

Results of the Visscher-Bowman Test in Urine from Non-pregnant Women, Pregnant Women and Puerperal Patients.

Type of Patient.	Total Tests.	Type of Result.						
		Negative Result.	Hazy.	Slight Fine Dark Brown Precipitate.	Heavy Fine, Dark Brown Precipitate.	Slight Brown Gelatinous Precipitate.	Dark Brown Gelatinous Precipitate.	Heavy Dark Brown Gelatinous Precipitate.
		%	%	%	%	%	%	%
Non-pregnant group (women 45 years of age and over)	58	30	14	16	5	16	17	3
Pregnant women: (a) Three months or less	91	16	8	14	3	24	29	6
(b) Later pregnancy ..	83	10	11	12	1	28	23	16
Puerperal patients ..	44	5	7	18	16	30	18	7
Total	276							

precipitate; (vi) dark brown gelatinous precipitate; (vii) heavy, dark brown gelatinous precipitate.

Types (v), (vi) and (vii) are the only types in which a definite reaction could be specified, and under such restrictions there were 36% positive reactions among 58 non-pregnant women, 59% positive results among 91 women with pregnancy of not longer than three months' standing, 67% positive results among women with pregnancy of longer than three months' duration, and 55% positive results among the 51 puerperal patients. The incidence of positive results in the pregnant groups is lower than reported by other workers and the number of positive results in the non-pregnant group is much higher.

Five specimens of urine from male persons were examined. Two gave negative results, two gave slight precipitates, and a pronounced reaction was obtained with the fifth specimen. In the last-mentioned case the specific gravity was 1030 and the urine produced marked reduction of Fehling's solution.

The high percentage of incorrect results among both pregnant and non-pregnant patients and the difficulty in deducing the nature of the precipitate obtained in tests giving positive results, from the curious mixture of chemical reagents used, led to doubt as to whether this precipitate really indicated the presence of the anterior pituitary hormone.

The following experiments were devised to test whether similar precipitates could be produced by other urinary constituents.

Effect of Concentration of Urine on the Visscher-Bowman Reaction.

Concentration of urine from male patients in which a negative response to the Visscher-Bowman test had been obtained resulted in definitely positive results being recorded. The same effects were noted when negative results had previously been obtained among pregnant and non-pregnant patients. Similar observations have been recorded by Wagner,[8] who proved that concentration increased the number of positive results and dilution the number of negative results, irrespective of whether the urine was obtained from pregnant or non-pregnant women or from males.

Effect of Glucose or Lactose on Visscher-Bowman Reaction.

Because of Dolff's statement[9] that reducing substances in the urine led to false positive Visscher-Bowman reactions, and because of the fact that glucose in the urine is frequently one of the earliest signs of pregnancy and lactose is often present in urine of patients after delivery, the samples of urine tested for the Visscher-Bowman reaction were also submitted to the Benedict test. This latter test was modified so as to detect smaller amounts of reducing sugar in the urine by the use of eight drops of urine to two cubic centimetres of Benedict solution. The mixture was then heated in a boiling water bath for ten minutes. The results obtained are tabulated in Table II. It is interesting that 58% of the positive reactions were associated with the presence of reducing sugar in the urine. This is particularly noticeable in the puerperal group in which 39 out of 51 tests showed this association.

Glucose was added to urine previously found to give negative results and the specimens were submitted to the Visscher-Bowman and Benedict tests. The urine was then incubated with yeast for twelve hours and the tests were repeated on the filtrate from these treated samples of urine. The results of typical experiments of this type are shown in Table III.

Twenty-two specimens of urine giving a positive Visscher-Bowman reaction and a positive response to the Benedict test were incubated with yeast for twelve hours and then retested. The results may be summarized as follows.

1. In five specimens the results of both the Visscher Bowman and the Benedict tests were negative after treatment with yeast.

2. In ten specimens the Visscher-Bowman produced a positive and the Benedict test a negative response after treatment with yeast.

3. In seven specimens the Visscher-Bowman and the Benedict tests both produced a positive response after incubation with yeast.

These experiments indicate that while glucose may give positive results in the Visscher-Bowman test without the presence of any pregnancy

Table II.

A Comparison of the Results of the Visscher-Bowman Test and the Benedict Test among Non-pregnant, Pregnant and Puerperal Patients.

Visscher-Bowman Test.	Benedict Test.	Non-pregnant Patients.	Puerperal Patients.	Early Pregnancy.	Late Pregnancy.
Positive.	Positive.	19	39	39	49
Positive.	Negative.	17	9	3	22
Negative.	Positive.	5	2	0	6
Negative.	Negative.	17	1	17	8
Total number of cases	258	58	51	59	85

TABLE III.

A Comparison of Visscher-Bowman and Benedict Reactions in: (a) Untreated urine; (b) this urine treated with glucose; (c) this urine treated with yeast after addition of glucose.

Urine Number.	(a) Original Urine.		(b) Urine *plus* Glucose.		(c) Urine *plus* Glucose Incubated with Yeast for Twelve Hours.	
	Visscher-Bowman Test.	Benedict Test.	Visscher-Bowman Test.	Benedict Test.	Visscher-Bowman Test.	Benedict Test.
50	Trace hazy precipitate.	Negative.	Positive.	Positive.	Trace.	Negative.
53	Trace hazy precipitate.	Negative.	Positive.	Positive.	Trace.	Negative.
D	Negative.	Negative.	+++	+++	Negative.	Negative.

hormone, other substances not fermentable by yeast may also be the cause of such reactions; for example, lactose in the puerperal cases.

These results are in agreement with the conclusion of Ostádal[9] that positive Visscher-Bowman reactions were obtained when urine contained lactose or other reducing carbohydrate.

Effect of Uric Acid and Urates on the Visscher-Bowman Test.

Uric acid dissolved in sodium hydroxide was added to three specimens of urine which gave negative responses to the Visscher-Bowman test. On repetition of the test fine dark brown precipitates were obtained.

A specimen of urine containing a heavy precipitate of amorphous urates was subjected to the Visscher-Bowman test and even after filtration a dark brown gelatinous precipitate was obtained.

Effect of Urinary Pigment on the Visscher-Bowman Test.

Sheehan[5] states that concentrated solutions of urinary pigments become intensely dark in colour, producing a melanin-like precipitate, when boiled for a few minutes with concentrated hydrochloric acid. He also showed that aqueous solutions of œstrin did not give the Visscher-Bowman reaction. He therefore holds that the positive results of the test are due to the effect of heat and concentrated hydrochloric acid on the normal urinary pigment urochrome.

In the present series of tests many specimens of urine very pale in colour gave very strong Visscher-Bowman reactions, while some deeply pigmented specimens gave very slight reactions or no reactions at all. An analysis of the results in this regard are correlated in Table IV.

The pigment theory therefore does not explain the reason for all of the positive reactions.

The Effect of Albumin on the Visscher-Bowman Test.

Wagner[3] states that albumin affects the Visscher-Bowman test, but gives no details of its action. In the present series many of the patients in the late pregnancy group were albuminurics. The albumin would be precipitated by the action of heat in the acid solution in the Visscher-Bowman test as a gelatinous mass, which with the dark coloration due to the action of the acid and heat on the urinary pigment could produce false positive results.

In regard to faintly coloured urine another explanation must be sought. Such urine giving a negative response to the Visscher-Bowman test was

TABLE IV.

A Comparison of Visscher-Bowman Test with Colour of the Urine.

Urine Colour.	Type of Result.						Total Tests.
	Negative Result.	Slight Fine Precipitate.	Fine Precipitate.	Slight Gelatinous Precipitate.	Dark Brown Gelatinous Precipitate.	Heavy Gelatinous Precipitate.	
Very pale yellow	11	2	4	8	3	4	32
Yellow	2	1	1	9	5	—	18
Deep yellow	1	—	1	1	8	2	13
Urobilin	1	—	—	—	3	—	4
Total tests	15	3	6	18	19	6	67

treated with glucose and albumin. The resultant mixtures were tested for specific gravity, albumin, Visscher-Bowman reaction and reduction by the Benedict test. The results of these tests are correlated in Table V.

Albumin, Benedict and Visscher-Bowman tests were performed on 26 specimens of urine, of which 10 were almost colourless. Four of the colourless specimens gave positive results to all tests. In one instance there was no albumin, very slight reduction and a very slight Visscher-Bowman reaction. In three specimens there was a very slight trace of albumin, very slight reduction and the Visscher-Bowman response was negative.

These results indicate that in urine containing albumin and reducing substances the possibility of false Visscher-Bowman reactions cannot be excluded.

Extraction of Prolan from Urine and its Relation to the Visscher-Bowman Reaction.

By treating urine from pregnant women with absolute alcohol as described by Rudd[10] the hormone could be obtained in concentrated form in water solution. This procedure was carried out on 25 specimens of urine, including those from 19 women whose pregnancy was of less than three months' duration, four from patients near term and two from patients in the puerperium.

The results of the Visscher-Bowman test on these urines before and after concentration are summarized as follows: (i) Eight specimens gave the same amount and type of precipitate in both instances. (ii) Two specimens showed greatly decreased precipitate after concentration. (iii) Seven specimens gave a negative response to the Visscher-Bowman test after concentration. (iv) Six specimens showed a slight white gelatinous precipitate after concentration. (v) In two specimens the results before and after concentration were negative.

In 16 of the 25 specimens definite reduction occurred with Benedict solution. These were associated with definite Visscher-Bowman reactions before treatment, and in 10 of the 16 positive reactions occurred after treatment. These results are probably due to adsorption of the glucose or lactose on the precipitate obtained by addition of alcohol. In those cases in which the result of the Benedict test was negative the Visscher-Bowman test also gave a negative response. These observations agree with the statement of Ostádal[9] that extracts of the hormone of the anterior lobe of the pituitary prepared from the urine of pregnant women failed to give positive Visscher-Bowman reactions.

Discussion.

From these observations correlated with the reports of other workers it is evident that the Visscher-Bowman reaction is not an indication of the presence of the hormone of the anterior lobe of the pituitary in urine of pregnant women. The reaction may be due: (*a*) to the presence of reducing carbohydrates, for example, glucose, particularly in the presence of albumin; (*b*) to marked concentrations of urinary pigment, with greater possibility of positive reaction in the presence of albumin; and (*c*) to the presence of other reducing substances in urine.

Conclusion.

The Visscher-Bowman test for pregnancy does not reveal the presence of the hormones of the anterior lobe of the pituitary present in the urine of pregnant women.

Acknowledgements.

I am greatly indebted to Sister Couper, of the Women's Hospital, for her cooperation in obtaining specimens for the tests and to Dr. Rome for his courtesy in checking data establishing the length of pregnancy.

Table V.

The Effect of the Addition of Glucose and Albumin to Urine on the Result of the Visscher-Bowman Test.

	Urine.	Specific Gravity.	Benedict Test.	Albumin.	Result of Visscher-Bowman Test.
Specimen 1 ..	Pale yellow	1006	Negative.	Trace.	Very slight gelatinous precipitate.
	Pale yellow *plus* glucose to concentration 0·5%	1008	++	+	Dark brown gelatinous precipitate.
	Pale yellow *plus* glucose to concentration 1·0%	1010	Complete reduction.	++	Dark brown gelatinous precipitate.
Specimen 2 ..	Pale yellow	1004	Negative.	Negative.	Negative.
	Pale yellow *plus* glucose to concentration 0·5%	1008	++	+	Dark brown gelatinous precipitate.
	Pale yellow *plus* glucose to concentration 1·0%.	1010	Complete reduction.	++	Increased amount dark brown gelatinous precipitate.

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CLINICAL DIETETICS.[1]

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DIETETICS is a big subject. Even had I the expert knowledge, it would not be possible to deal comprehensively with it in one lecture. Under the circumstances, I must be content with trying, as Dr. Castle, of Boston, put it here recently, to "hit the high spots". I shall discuss a little series of questions: (i) What constitutes a food? (ii) What happens to the food we eat? (iii) What are the normal food requirements of people in good health? (iv) In what way can the knowledge of dietetics be applied in the treatment of human diseases?

What Constitutes a Food?

Very few articles of diet are simple chemical substances. Most are mixtures of more or less complex materials: (i) proteins, for example, casein, albumin, myosin, gelatine, gluten; (ii) fats, for example, triglycerides of fatty acids, such as butyric, oleic, palmitic, stearic; (iii) carbohydrates, for example, sugar, starch, dextrin, glycogen, cellulose; (iv) mineral matters, for example, common salt, compounds of calcium, iron, iodine, potassium, magnesium *et cetera*; (v) extractives and flavouring agents; (vi) vitamins; (vii) water.

The nutritive constituents of food in accordance with their function in the body can be conveniently classified into tissue formers and energy producers. The tissue formers are complete proteins, incomplete proteins in the presence of complete proteins, mineral matter, and water. The work and heat producers are complete and incomplete proteins, fats and carbohydrates. Mineral matters and vitamins act as catalysers.

Later on, I shall discuss complete and incomplete proteins more fully. The point to remember at the moment is that food is more than just proteins, fats and carbohydrates. It is now an old story in the history of dietetics that a mixture of pure proteins, fats and carbohydrates given experimentally to animals failed to sustain healthy life. And so came the discovery of vitamins. But the history of vitamins, fascinating though it is, is a long story, and it has been so recently told us by Dr. Castle that I shall say no more about it. We can just note that a diet is inadequate if it lacks a fair proportion of the so-called protective foods. These contain ample amounts of the several vitamins, and are mainly: (i) milk and milk products, such as butter, cream and cheese; (ii) fresh vegetables and fruits, especially green salad vegetables and the yellow vegetables and fruits; (iii) liver, including fish liver and fish-liver oil; (iv) fish, especially the fat fish and fish roes; (v) eggs.

I shall tell tonight the older story of the nutritive value of the various foodstuffs and of this business of Calories. The Calorie of the dietitian is the large Calorie—Calorie with a capital C—and it indicates the amount of heat energy required to raise the temperature of a litre of water by 1° C. The little calorie of the physicist is a thousandth part of this. The energy value of the common foods is listed in most of the medical text-books; a working knowledge of the constituents of ordinary foods is obviously essential in the rational practice of dietetics.

What Happens to the Food We Eat?

We submit our food to a number of complicated processes. These include: (i) ingestion, (ii) digestion, (iii) absorption, (iv) anabolism or assimilation, (v) katabolism and (vi) excretion of waste.

Lawrence, in his book on diabetes, produces a useful diagram showing the details of the changes the three great food constituents go through (see Figure I).

The proteins are highly important. Without them growth and tissue repair are impossible. In the process of digestion the proteins are resolved into amino-acids. The molecular weight of the typical protein is very high, and the protein molecule yields a large number of amino-acids, generally from 12 to 20. Analyses of various proteins show that the percentages of individual amino-acids vary greatly in different proteins. Table I, reproduced from Sherman's "Chemistry of Food and Nutrition", shows the relative amounts of amino-acid radicles constituting four common proteins. Casein of milk is a complete protein, the others are not. Note especially the zein of maize has no lysine nor tryptophane radicle.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 24, 1938.

TABLE I.

Comparative Composition of Proteins: The Amino-Acid Percentages Yielded on Hydrolysis. (After Sherman.)

Amino-Acid.	Percentage Yielded on Hydrolysis.			
	Casein.	Gelatin.	Gliadin.	Zein.
Glycine	0·5	25·5	0·5	0·0
Alanine	1·8	8·7	2·0	9·8
Valine	6·7	1·0	3·3	1·9
"Leucine"	9·7	7·1	6·6	25·0
Proline	8·0	9·5	13·2	9·0
Hydroxyproline	0·23	14·1	?	?
Phenylalanine	3·9	1·4	2·4	7·6
Glutamic acid	21·8	5·8	43·7	31·3
Hydroxy glutamic acid	10·5	0·0	2·4	2·5
Aspartic acid	4·1	3·4	0·8	1·8
Serine	0·5	0·4	0·13	1·0
Tyrosine	6·5	0·01	3·5	5·9
Cystine	0·34	0·31	2·1	1·0
Histidine	1·8	0·9	2·4	0·8
Arginine	3·8	8·2	3·1	1·8
Lysine	6·3	5·9	0·9	0·0
Tryptophane	2·2	0·0	1·14	0·0
Ammonia	1·6	0·4	5·2	3·6

The crux of the matter is this. The nutritive value of food proteins depends essentially on the kinds of amino-acid radicles which they contain, and the quantity of each radicle they contain.

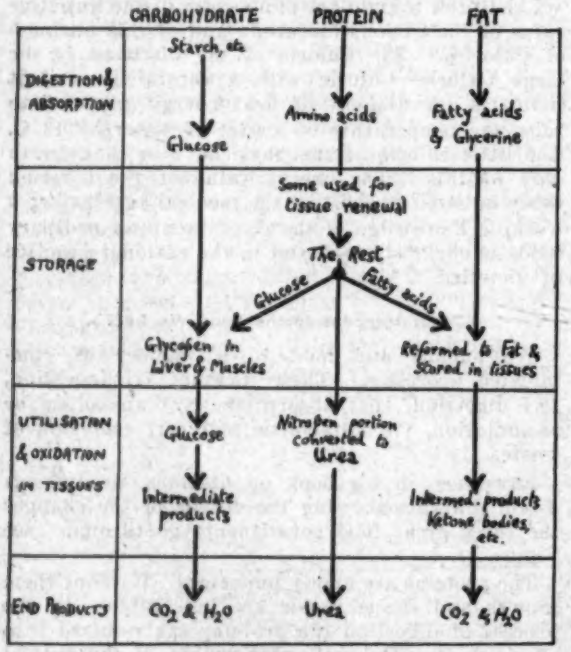

FIGURE I.

Illustrating the metabolic changes undergone by each of the three great classes of foodstuffs. (Modified after R. D. Lawrence.)

Sherman gives a useful way of grouping proteins from this standpoint:

A. "Complete": Maintaining life and providing for normal growth of the young when used as a sole protein food. Examples of these are: casein and lactalbumin of milk; ovalbumin and ovovitellin of egg; glycinin of soy bean; excelsin of Brazil nut; edestin, glutenin and maize glutelin of the cereal grains.

B. "Partially incomplete": Maintaining life, but not supporting normal growth. Gliadin of wheat is a well demonstrated example of this class. Hordein of barley and the prolamin of rye are similar in nutritive value to gliadin.

C. "Incomplete": Incapable either of maintaining life or of supporting growth, when fed as the sole protein. Zein of corn (maize) and gelatine are the conspicuous examples.

Experiments cited by Sherman clearly illustrate the method of elucidating this information. The experiments indicate the growth of rats whose protein intake was carefully restricted along specialized lines. The animals had only one protein fed to them, and had a standard diet of fats and carbohydrates. Figure II, constructed from

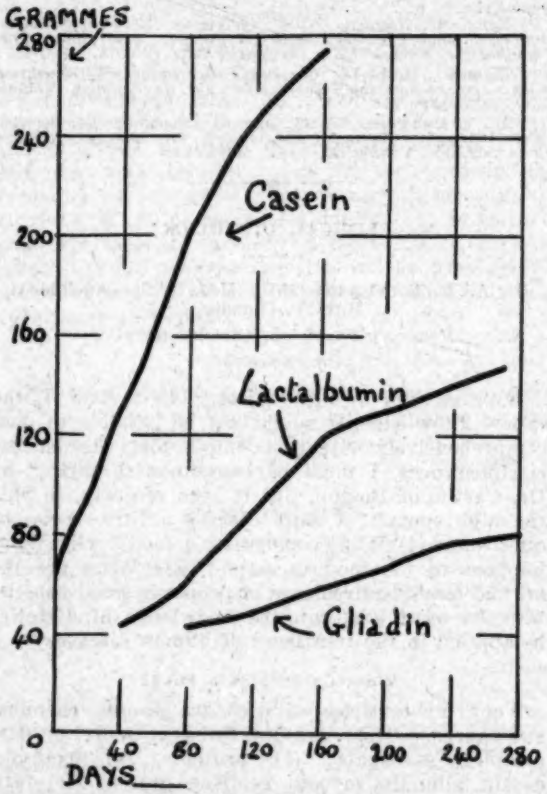

FIGURE II.

Illustrating the effect of feeding rats with a diet containing standard amounts of fat and carbohydrate, but only one protein material. The figure is a composite one, adapted from Mendel (*vide* Sherman).

Sherman's data, shows that the growth of the rat was best when the protein given was casein, fairly good when it was lactalbumin, and poor when it was gliadin. When the protein was zein, the rat, far from flourishing, just began to wilt (Figure III). You will remember that zein lacks the amino-acid radicle tryptophane. When tryptophane was added to the diet the rat maintained its condition, but did not grow. When lysine was added as well—now remember that zein is lacking also in lysine—the rat grew satisfactorily. The deductions are:

(i) casein and lactalbumin are complete proteins; (ii) gliadin is a partially incomplete protein; (iii) zein is an incomplete protein; (iv) zein with tryptophane serves to maintain life in the same way as a partially incomplete protein does; (v) zein with tryptophane and lysine serves to maintain life and support growth in the same manner as a complete protein. (Tryptophane and lysine radicles are not present in zein, and the lack of them renders zein itself a poor protein.)

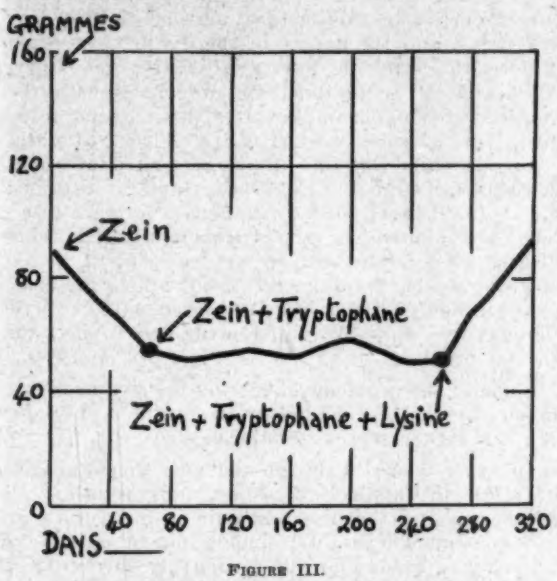

FIGURE III.

Showing the effect of feeding a rat with a diet containing zein as its only protein constituent. The animal wasted. On the addition of tryptophane the weight remained stationary. The further addition of lysine gave the rapid increase of growth as occurs when "complete" proteins are given. The figure is from Mendel's work, quoted by Sherman.

What are the Food Requirements in Health?

What should be the average man's diet expressed as energy value? Scores of analyses by various investigators of the freely chosen diets taken by healthy adult males, neither gaining nor losing weight, show the value at about 3,000 Calories a day. In round figures, the daily intake of the ordinary man is as follows:

Proteins	100 grammes
Fats	100 grammes
Carbohydrates	300 to 500 grammes

How much protein is required? The jealous guarding of the nitrogen equilibrium makes it difficult to decide. Hutchison and Mottram sum up the matter by saying that the amount of protein in the diet should not fall below 80 grammes a day, and that at least 5% of the total Calories should be supplied by first-class protein. This required amount of animal protein is contained in eight ounces of beef, in five or six eggs, or in a quart of milk.

These figures roughly represent the food value of the everyday diet of you and me. In England, a British Medical Association committee has had a special cookery book prepared. It is designed to assist the housewife of the ordinary home to select wisely and to cook suitably the food for her family.

This average daily intake of the ordinary healthy man gives only a rough basis of dietary needs. It is probably true that dietary standards reflect habits rather than actual needs. The scientist begins his investigation of the actual food needs with a study of basal metabolism. The surface area, the skin area, is a big factor. It has been found that in animals so different as dogs, horses, mice and men, the output of Calories per square metre of body surface is about 1,000 per day. This, as Hutchison and Mottram say, is surely something fundamental. It seems a constant for a group at least of the warm-blooded animals. With a knowledge of the subject's surface area, we can proceed to compute his food needs. Now to measure the area of a man's skin is no easy job, and tables and graphs have been constructed so that the area may be deduced from a knowledge of the height and weight. The chart used in the Mayo Clinic is helpful and is easy to handle.

The requirements for total metabolism, the energy requirements for ordinary active life, are represented by a figure about twice that for the resting or basal metabolism. The influence of sex and age and work on food requirements must be noted. Women require about 8% less than men. Once a person has reached adult life, the food requirements remain nearly a constant, but they fall gradually with the approach of senility. The requirements of men in different occupations are shown in Hutchison and Mottram's book, as follows:

	Calories (Net).
Tailor	2,500
Bookbinder	2,800
Shoemaker	2,850
Metal worker	3,200
Carpenter	3,200
Painter	3,250
Stonemason	4,400
Woodcutter	5,000

Tables are constructed showing the requirements of childhood. That of Cathcart and Murray (quoted by Hutchison and Mottram) is an example. The coefficients in the table show the energy value of the child's diet relative to that of the average adult male (see Table II).

TABLE II.

Caloric Requirements in Childhood, according to Cathcart and Murray, as quoted by Hutchison and Mottram.

Age in Years.	Utilizable Calories.	Coefficients.
0 to 1	600	0·2
1 to 2	900	0·3
2 to 3	1,200	0·4
3 to 6	1,500	0·5
6 to 8	1,800	0·6
8 to 10	2,100	0·7
10 to 12	2,400	0·8
12 to 14	2,700	0·9
Girls 14 and on	2,500	0·83
Boys 14 and on	3,000	1·00

Even when a child is at rest, his vigorously growing tissues exhibit a much more active metabolism than the mature tissues of the adult. The boy of fifteen needs 45 Calories per square metre per hour; at the age of twenty years the need has been reduced to 40 Calories; and at 60 years only 35 Calories are needed. In any case, weight for weight, the child's food need is greater than the adult's, owing to the surface area factor. It is also to be noted that persons of different temperament vary in their requirements. Hutchison and Mottram give a chart to illustrate this point; I have reproduced portion of it as Figure IV.

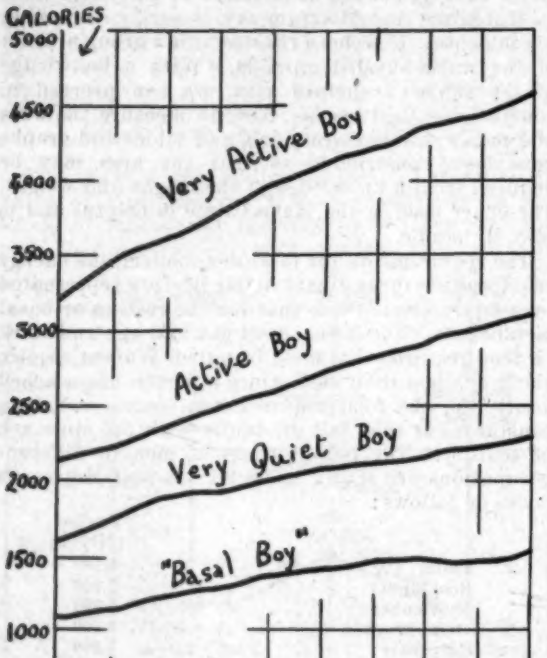

FIGURE IV.

Showing the caloric requirements of boys of different ages and temperaments. (From Hutchison and Mottram.)

These scientific considerations have their value; but in actual practice "seeing is believing". The best indications as to the sufficiency of a normal person's diet are that it satisfies hunger, maintains good nutrition, and keeps a satisfactory weight level. But it is useful to remember that the work we do, the games we play, and even the thoughts we think, have a material influence on our food needs.

The Diet in Disease.

A proper understanding of the principles underlying the normal diet of healthy people is necessary in a consideration of diets for the sick. The diet of a person often must be modified when he becomes sick. The physical character of the foodstuffs; their digestibility; the energy value of the diet; the relative amounts of protein, carbohydrate and fat; the relative amounts of acid and base materials, and the amount of fluids—all these factors have to be considered when dietaries are being formulated for the sick.

The processes following the intake of food, discussed earlier in this lecture, may be disturbed at any point, and the nature of the disturbance often decides the type of dietary alteration necessary. An attack of acute tonsillitis interferes with the swallowing of food and requires the material to be given in a soft, non-irritating form. Palatal paralysis calls for tube feeding; gastro-intestinal diseases interfere with digestion and absorption; metabolic diseases, such as diabetes and gout, interfere with anabolism and katabolism; and renal disease hinders the satisfactory removal of breakdown products. In all such cases, in which the basic processes of nutrition are interfered with, a little thought will show the clinician the way to meet the special need.

Some of the problems of dieting the sick are more involved, and I review briefly the suitable plans for dealing with certain special cases.

In acute fevers, although the nutritional need is high, the processes of digestion and excretion are hampered, and the need for fluid is increased. In any continued fever, it is impossible to satisfy the nutritional need, and the patient is sure to lose weight. The situation can best be met by the provision of ample fluids, mainly as milk, alone or with egg, fruit juices with glucose, and thin concoctions of cooked cereals.

In wasting diseases, or in convalescence from debilitating illnesses, a high caloric diet, easy of digestion, must be given. The following is a suitable programme giving a food value of about 4,000 Calories:

Breakfast: Fruit juice or grapefruit; cereal with fruit and cream; one or two eggs, soft-cooked; two slices of toast with butter and jam; and coffee with cream and sugar: 1,030 Calories.

Morning Tea: Milk, with cream: 300 Calories.

Lunch: Fish or chicken, and salad; baked apple with cream; bread, butter and cakes: 1,100 Calories.

Dinner: Cream soup and rusks; roast lamb, potato, peas *et cetera*; light pudding: 1,300 Calories.

Supper: Milk; "Ovaltine": 300 Calories.

When the clinical problem is the reduction of obesity, it is obvious that a low caloric intake is necessary, and that increased exercise to promote increased metabolic activity will also serve a useful purpose. Too rapid loss of weight must be guarded against, and excessive exercising of the obese has its risks. A suitable dietary plan, based on that put forward by von Noorden years ago, is here shown. This has a low caloric value, approximately 1,000 Calories, and a high satiety value. The giving of

frequent small meals obviates the feeling of hunger. The table is as follows:

Breakfast: Half a small orange or grapefruit; a thin slice of toast; coffee with skimmed milk and water.

Morning Tea: A glass of skimmed milk and two biscuits.

Lunch: One egg or a small helping of lean meat; vegetables of low caloric value; a tiny square of butter; a small helping of plain dessert; tea.

Afternoon Tea: A glass of skimmed milk and a biscuit.

Dinner: Clear soup; a small helping of lean meat, with vegetables of low food value; a small slice of bread; tea or black coffee.

Supper: A glass of skimmed milk; a biscuit; lettuce.

For patients with severe degrees of cardiac failure, I have found the Karrell diet suitable. As cardiac efficiency returns, the diet is gradually increased; but large meals of bulky, indigestible foods must be avoided. The main factors are a moderately low caloric value, a low protein intake, but an ample supply of easily assimilated carbohydrates.

In feeding victims of hyperpiesia, I have often found that a suitable diet relieves symptoms, especially if the patient is greatly overweight. In such cases a diet programme consisting of 50 grammes of protein, 50 grammes of fat, 140 grammes of carbohydrate, and a total caloric value of about 1,200, is suitable. The fluid intake should not exceed 40 ounces a day. The following is the type of programme I often use in such cases:

Breakfast: Orange; bread and butter; a cup of weak tea.

Lunch: One egg or one ounce of cheese; bread and butter; vegetables poor in carbohydrate, such as salad; six ounces of milk.

Dinner: Small helping of fish or meat, with vegetables; fruit; six ounces of milk; light pudding.

Supper: Fruit and milk.

Dieting in nephritis presents its special difficulties. In acute nephritis the Karrell type of diet is suitable for the first few days. As improvement follows there may be added fruit juices, cooked fruit, ripe banana, cooked cereals, toast or bread, cream, unsalted butter, the total giving up to a value of 1,800 Calories per day, with the protein at about 25 grammes. In chronic nephritis with œdema a similar diet is suitable, but additional protein in the form of lean meat and eggs will be necessary. The old idea that eggs should be avoided by patients with albuminuria should surely be completely abandoned.

In nephrosis, the Epstein diet, giving a protein value up to two grammes per kilogram of body weight, is often useful in promoting diuresis. The intake of fats, salt and fluids is kept low, and the diet consists largely of eggs, lean meat, skimmed milk, fruit and cereals. The value of a high protein diet in nephrosis depends on the long-recognized specific dynamic action of protein, and the increased metabolic activity it stimulates.

There are numbers of other special dietary programmes that might be reviewed. There is, for instance, the use of diet to influence—as far as it can be influenced—the hydrogen-ion concentration of the blood and tissue fluids. The giving of acid-forming foods has become rather popular in recent years in the treatment of sufferers from epilepsy, asthma and pyelitis. The acid-forming foods include eggs, meat, fish, poultry, bread, and other cereal foods, pastries, puddings and cheese. The main base-forming foods are most fruits, except plums; vegetables, especially spinach; milk and nuts.

There has been a good deal of interest lately in the mineral constituents of the dietary, in such matters as calcium metabolism and its influence on dental development. The effect of low calcium intake in lead poisoning was studied by Aub and Donald Hunter. The idea is that lead in plumbism is stored in the bones, and that, when a negative calcium balance is induced by means of a calcium-poor diet, there is increased excretion of lead along with the high calcium excretion. The decalcifying process brings about "deleading" of the body. Foods rich in calcium must be omitted from the "deleading" diet. These calcium-rich foods are milk and cheese, eggs, green vegetables, most root vegetables, dried fruits and legumes. Such a programme is suitable only for a short time, and must be used with caution.

I have given but a few examples of treatment by dieting, and these only in a general way. After all, it is the general principles that we as clinicians need to grasp and apply, rather than detailed and inflexible programmes.

And now a final question:

Is Dieting Really Important?

So many fads and cults have grown up involving food habits that the ordinary man may well wonder what the correct views really are. If he reads such recent American books as "A Hundred Million Guinea Pigs", or "Eat, Drink, and be Wary", our ordinary man will become even more perplexed. There are so many special schemes and "stunts". There is, for instance, the Hay system. The sponsors of this system do not suggest that we should take to grass-eating, like Nebuchadnezzar of old, but they do say that the taking of proteins and carbohydrates together at meals is harmful. What an absurdity this is needs no further comment.

The cult of vegetarianism is fairly common. The recent article by Sir Leonard Hill in *The British Medical Journal* excited much interest. In this connexion, I agree with Rabinowitch that, if the small boy whose diet programme was discussed by Sir Leonard Hill adhered strictly to that diet, the lad would "disappear completely in less than two years". In any case, the addict to vegetarianism generally takes milk and eggs, and so gets enough good animal proteins and Calories to satisfy his needs.

And then there is the commercial aspect of food. Vitamin deficiency has been studied fully by the scientists, and the food manufacturers have been quick to use, or rather misuse, the new knowledge for commercial profit, at the cost of public peace of

mind. The value of mineral matters in the diet and the effects of their reduction on health are also in the limelight. We read in our newspaper advertisements of "mineral starvation". Body growth and dental caries are much talked of. This problem of teeth decay is a difficult one. I wonder whether we really understand why teeth decay any more clearly than we understand why heads go bald.

Of the peculiar diets supposed to be "good for rheumatism" there is no end. The use of lemons is much argued about and the cult of "acid poisoning" dies hard. And so we could go on discussing this matter of food.

To sum it up, as far as the general food arrangements of the community are concerned, we may say that the greatest modern danger is the sophistication of foodstuffs, and the greatest modern need is simplicity. We might even do well to remember, as a distinguished colleague of mine has put it, that our great Empire was built up, not on vitamins and Calories, but on beer and beef.

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H. C. Sherman: "Chemistry of Food and Nutrition", 1937.

THE VASCULAR CONDITIONS OF MUSCULAR EXERCISE.

By N. D. ROYLE, M.D., Ch.M., F.R.A.C.S.,
Sydney.

IT is asserted by physiologists that a dilatation of arterioles accompanies muscular contraction, and that there is a fall in peripheral resistance in the vascular system.[1][2] It is known that the capillaries of muscles are dilated during exercise, and if the arterials are also dilated, a wrestler in action would have practically no diastolic pressure—a condition that would speedily put him out of action.

It is improbable that the capillaries and arterioles are ever dilated at the same time, except as a local condition in acute inflammation, as the effect of a toxin, or under the influence of large doses of certain agents, such as histamine or ether.

There appears to be a reciprocal arrangement between the capillaries and arterioles, so that when one is contracted, the other is dilated. I have called this the arteriolar-capillary reaction,[3] and it is well exemplified in the vascular conditions of exercise.

The subjects in most of these experiments were medical students about twenty years of age. The readings were taken by the auscultatory method and a mercury sphygmomanometer was used.

Experiments.

The following experiments were carried out:

1. The blood pressure was measured in the exercised limb immediately after exercise. The results are recorded in Table I.

TABLE I.

Systolic and Diastolic Blood Pressure Before Exercise in Millimetres of Mercury.	Systolic and Diastolic Blood Pressure After Exercise in Millimetres of Mercury.	Increase in Blood Pressure in Millimetres of Mercury.	
		Diastolic.	Systolic.
L.U.L.			
88–122	98–124	+10	+2
90–118	96–130	+6	+12
78–118	78–130	+0	+12
74–120	80–132	+6	+12
76–124	76–128	+0	+4
72–134	80–140	+8	+6
72–122	80–130	+8	+8
78–110	86–120	+8	+10
78–132	86–132	+8	+0
86–134	90–140	+4	+6
70–114	74–128	+4	+14
74–118	80–134	+6	+16
78–120	86–130	+8	+10
80–120	86–130	+6	+10
78–110	90–124	+12	+14
76–112	92–135	+16	+23

From this table it will be seen that the diastolic pressure rose in every experiment excepting two. This shows that the peripheral resistance, instead of being decreased, was increased.

2. Table II shows the changes in blood pressure in the contralateral limb during exercise of one upper limb. The method of exercise (in 1 and 2) was to compress a grip dynamometer as strongly as possible. It will be seen that there is a great rise in diastolic pressure and therefore in peripheral resistance in the contralateral limb in spite of the dilatation of capillaries.

TABLE II.

Changes in Blood Pressure of Contralateral Limb During Exercise in One Upper Limb.

Subject.	Before Exercise. Diastolic and Systolic Blood Pressure.	After Exercise.		
		Diastolic and Systolic Blood Pressure.	Increase.	
			Diastolic.	Systolic.
A	84–126	102–154	+18	+28
B	88–122	108–142	+20	+20
C	78–118	101–148	+23	+30
D	74–120	110–145	+36	+25
E	76–124	104–154	+28	+30
F	72–134	96–160	+24	+26
G	76–112	102–132	+26	+20
H	72–132	84–148	+12	+16
I	88–154	114–	+26	+

3. The effect of moderate and strenuous exercise on diastolic pressure is shown in Table III. It will be seen that the diastolic pressure rose with the strenuousness of the exercise.

TABLE III.

Subject.	Diastolic and Systolic Blood Pressure of Left Upper Limb, During Exercise of Right Upper Limb.			Increase in Diastolic and Systolic Blood Pressure.	
	Before Exercise.	Moderate Exercise.	Strenuous Exercise.	After Moderate Exercise.	After Strenuous Exercise.
A	72–132	84–148	114–154	12–16	42–22
B	48–105	90–124	100–152	42–19	52–47
C	80–112	88–122	98–132	8–10	18–20

The moderate exercise was compression of a grip dynamometer, and the strenuous exercise was lifting of a twenty-eight pound weight from the shoulder to the position of extension of the right upper limb above the head. This, of course, involved more muscles than those of the upper limb.

4. The effect of exercise of both upper limbs on the blood pressure of the left lower limb is shown in Table IV.

TABLE IV.

Diastolic and Systolic Blood Pressure in Millimetres of Mercury in Left Lower Limb During Exercise of Both Upper Limbs.

Subject.	Before Exercise.	Strenuous Exercise.
A	68–130	100–154
B	84–124	104–168
C	86–150	180–220

The subject lay face downwards and compressed a grip dynamometer as strenuously as possible with both hands. This action involved the muscles of both upper limbs as well as the muscles of the trunk. The subject, who registered a gain of ninety-four millimetres in diastolic pressure, made a particularly strenuous effort during the reading of the sphygmomanometer.

5. Table V shows the blood pressure in the left upper limb before and just after exercise of both lower limbs carried out on a bicycle ergometer against resistance. The last and second last two readings were in younger subjects and taken after each had peddled a player piano.

TABLE V.

Diastolic and Systolic Blood Pressure in Left Upper Limb Immediately After Exercise of Both Lower Limbs.

Subject.	Before Exercise.	After Exercise.	Three Minutes After.	Difference in Pressure.	
A	92–146	108–180	84–180	+16	+34
B	90–130	78–160		−12	+30
C	100–140	104–180		+4	+40
D	102–138	90–200	74–170	−12	+62
E	92–120	100–150	94–126	+8	+30
F	100–140	96–166		−4	+26
G	94–148	94–180		+0	+32
H	94–140	100–130		+6	−10
I	64–110	72–164	50–120	+8	+54
J	68–95	78–124	72–98	+10	+29

It will be noticed that in six subjects the diastolic pressure rose; in three there was a fall; and in one neither rise nor fall. Possibly the readings were not taken soon enough in the latter four instances.

6. When the reading in the left lower limb was taken during exercise of the right lower limb as seen in Table VI, the results were similar to those in the contralateral in the case of the readings in the upper limbs (Table II).

TABLE VI.

Diastolic and Systolic Blood Pressure in Left Lower Limb During Exercise of Right Lower Limb.

Subject.	Before Exercise.	During Exercise.	Difference in Pressure.	
A	120–185	140–200	+20	+15
B	120–170	150–175	+30	+5
C	110–150	125–160	+15	+10

Comments on these Experiments.

The findings in these experiments show that the diastolic pressure rises with the active contraction of muscles and that the degree of rise is related to the strenuousness of the exercise. It will also be seen that the exercise of the upper limbs is accompanied by a rise in diastolic pressure in the lower limbs, and the exercise in the lower limbs is accompanied by a rise in diastolic pressure in most subjects in the upper limbs. Exercise of both upper and lower limbs is accompanied by a rise of diastolic pressure in the contralateral lower or upper limb. I have a patient in whom the diastolic pressure is ten millimetres less in a wasted upper limb than in an hypertrophied opposite upper limb, and there is a difference of 23 millimetres in the diastolic pressure of the lower limbs. The blood pressure in the lower limbs with large muscles is higher than that in the upper limbs of the same subject with smaller muscles. Can it be that a local reflex operates through the vasomotor centres of the spinal cord, or that there is more force necessary to compress the muscles of the lower limbs? The local reflex was suggested by me in a paper entitled "The Surgical Treatment of Spastic Paralysis",[4] and renders possible the higher diastolic reading in spastic limbs.

It is also interesting to speculate on what would happen to the diastolic pressure if there were no muscular activity going on in the body. For example, I suggest that the low diastolic pressure during sleep is maintained by the contraction of the muscles of respiration: in other words, that the diastolic pressure is a manifestation of reflex action.

It is also interesting to note that in a state of activity of muscle the capillaries are dilated, and these experiments show that the arterioles are constricted more than usually. It is also known that in a state of rest the capillaries of muscle are contracted, and from these experiments it may be inferred that the arterioles are more dilated during muscular relaxation since muscular contraction increases the degree of constriction. The former condition of the vessels retards the rate of capillary flow and allows more time for oxygen to act; the latter state accelerates the rate of capillary flow and so gives less time for oxygen to act.

A correspondent of mine[5] suggested a pair of bellows as an illustration of the action of oxygen. The nozzle represents the diastolic pressure, the gradually retarded stream of air as it reaches and is impeded by the live coals represents the capillary flow. The increased combustion is the result of the action of oxygen.

This principle of vascular action must apply to all activity both in the nervous system and in glands, as it is shown by these experiments to apply to muscular activity.

The results of these experiments are also evidence against the theory of chemical transmission of nerve impulses. Acetylcholine is said to contract the capillaries and relax the arterioles, but, according to these experiments, the arterioles are contracted

to a greater degree during exercise. In this case the capillaries would be, and are in fact, dilated, for it is impossible in the normal subject to have both arterioles and capillaries constricted at the same time. Such a phenomenon is, I believe, seen in Raynaud's disease.

These experiments also give a reason for the practice of stretching indulged in by animals and some humans after repose. The movements involved in stretching raise the blood pressure, which has been lowered in rest.

Summary.

Muscular contraction raises the diastolic blood pressure (*a*) in the ipsilateral limb and (*b*) in the contralateral limb.

Exercise of the upper limb raises the diastolic pressure in the lower limb and *vice versa*.

The vascular conditions accompanying active muscle contraction are dilatation of capillaries and greater constriction of arterioles. The vascular conditions of muscles at rest are constriction of capillaries and dilatation of arterioles.

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(5) W. P. Kelly: THE MEDICAL JOURNAL OF AUSTRALIA, July 10, 1937, page 76.

Reports of Cases.

ARSENICAL POISONING TREATED BY ADSORPTION THERAPY.

By LOUIS E. S. GELLÉ, M.B., B.S. (Durham), *Perth*.

THE patient, a female, aged thirty years, who had been under previous treatment for *pruritus vulvæ*, was admitted to hospital on May 24, 1938, because of a suspicion of amœbic dysentery. One tablet of "Stovarsol" *per diem* was prescribed. This was taken for six days without untoward results, when the patient exhibited a purpuric rash on the face and limbs and the temperature rose to 38·9° C. (102° F.), and on the subsequent day to 39·9° C. (103·8° F.). These symptoms resembling those of a prevalent type of influenza, the "Stovarsol" was immediately stopped and appropriate treatment administered, including the injection of 4·0 cubic centimetres of "Edwenil", which brought the temperature down to 36·7° C. (98° F.) in the course of twelve hours. The temperature rose soon after to 37·8° C. (100° F.) and fluctuated between this temperature and 38·9° C. (102° F.). Suddenly it rose to over 39·4° C. (103° F.), the exacerbation being associated with severe bowel movement and the passage of large quantities of blood. On investigation, this was found to be due to the administration of a tablet of "Stovarsol" by a nurse who was new to the ward. The rash became generalized, the patient being lobster red and acutely ill; the temperature ranged between 37·2° C. (99° F.) and 38·9° C. (102° F.) for weeks on end, during all of June and July.

I am of the opinion that this case of very acute arsenical poisoning, which resulted from such a small dose of "Stovarsol" as one tablet per day for a period of hardly five or six days, could only have resulted from the intensely sensitive and inflamed condition of the mucous membrane of the bowel caused through prolonged and persistent use of Epsom salt, taken by the patient for months under the belief that frequent evacuation would improve her condition (*pruritus vulvæ* and *pruritis ani*, supposedly due to thread-worms), for which she had been in more or less constant treatment by a variety of medical men and hospitals in this and other States for a period of fifteen years. For the past three or four years she had drawn an invalid pension on this account.

Sodium thiosulphate was administered very early by way of antidote, but produced no alleviation of the patient's condition. The life of the patient was precarious. Previous knowledge of the value of kaolin in the treatment of moribund patients decided me to restrict the patient to "Kaylene" (ultra-fine colloidal kaolin) in doses of eight grammes (two drachms).

The patient was acutely ill and the temperature came down to normal only after eight weeks' treatment, by which time she had a paresis of arms and legs. During the whole of the term of convalescence, "Kaylene" was continued, and under efficient nursing she regained the use of her legs, but even now is still very weak. The whole surface of her body, including the scalp, became desquamated, and she lost considerable weight, this being aggravated by the fact that she would take milk only disguised as tea or "Bovril". The main source of nourishment was fruit juices.

This case demonstrates the value of the sustained administration of pure colloidal kaolin as a means of detoxication by adsorption and allaying by mechanical protection the inflammation of the mucosa. To this, without doubt, we owe the saving of the life of this patient.

Reviews.

OCCIPITO-POSTERIOR POSITIONS.

THE second edition of Dr. Beard's small book on "The Occipito-Posterior Position: Its Mechanism and Treatment" has appeared.[1] We reviewed the first edition in 1933. The edition which we have had submitted to us for review was published in 1937. The author has added a little new matter, the largest portion being in regard to the manual rotation of the trunk by internal manipulation recommended by Professor Bjornson, of Winnipeg.

The author divides the occipito-posterior cases into: (*a*) true occipito-posterior presentations in which the occiput is at the sacro-iliac joint, (*b*) the intermediate occipito-posterior presentation in which the occiput is in front of the sacro-iliac joint. He states that the true occipito-posterior presentation is relatively uncommon. He also states that in the intermediate occipito-posterior position the occiput coming in contact with the pubo-coccygeal portion of the *levator ani* is rotated to the front, whereas in the true occipito-posterior birth face-to-pubes is the commoner termination.

There is at the end of the book a long list of conclusions which could be abbreviated with benefit; some other minor alterations would make the arguments more easily understood.

There is no doubt that Dr. Beard has brought forward interesting observations and interesting deductions. His advice as regards to treatment is sound and his book is one that can be recommended to all who are concerned with obstetrics.

[1] "The Occipito-Posterior Position: Its Mechanism and Treatment, with a New Conception of the Role of the Pelvic Floor", by R. Beard, M.C., M.B., B.S., F.R.C.S., F.R.A.C.S., F.C.O.G.; Second Edition; 1937. Adelaide: F. W. Preece and Sons. Demy 8vo, pp. 39, with illustrations.

The Medical Journal of Australia

SATURDAY, MARCH 25, 1939.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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POST-GRADUATE EDUCATION IN NEW SOUTH WALES.

THE growth of facilities for post-graduate study in New South Wales has been reported from time to time in this journal. With the inauguration of the New South Wales Post-Graduate Committee in Medicine, formed by the adoption of a new by-law by the Senate of the University of Sydney after it had been approached by the erstwhile New South Wales Permanent Post-Graduate Committee, and with the passage of *The Prince Henry Hospital Act*, 1936, post-graduate teaching in New South Wales entered on a new and hopeful phase. The new committee was constituted on October 14, 1935, and the Prince Henry Hospital was opened as a post-graduate school on April 5, 1938. The hand-book announcing the post-graduate medical courses for 1939, recently issued, contains a great deal of valuable information. Attention should be drawn to some of the newer arrangements.

The most important innovation is the creation by the Board of Directors of the Prince Henry Hospital of fellowships in medicine and surgery. Senior and junior fellowships are being created in both subjects. These fellowships will be awarded every year by the Board on the recommendation of the Advisory Committee. They will be tenable for one year and the successful applicants, who will be chosen after advertisement, will reside at the hospital and be attached to the medical and surgical units. It will be remembered that last year Directors of the Medical and Surgical Units were appointed in the persons of Dr. S. A. Smith and Dr. H. R. G. Poate. Fellows will be provided with board and residence free of charge and the annual value of the fellowships will be £400 for seniors and £300 for juniors. Each Fellow will be required to give the whole of his time to the fellowship—no private practice will be allowed. Since they will be members of the staff of the hospital, Fellows will take an active part in the treatment of patients. A moment's consideration will make it clear that no post-graduate student in attendance at a hospital can be allowed, for example, to perform a surgical operation unless he is a member of the hospital staff. Nothing but approval can be given to the statement that Fellows will be chosen from graduates who intend to take a higher degree or diploma. We are also informed that they "may be selected for their promise of aptitude for investigating medical problems". We should have preferred the words to be "shall, if they are available, be selected . . ." The personnel of the Board being as it is, we may presume that, other things being equal, preference will be given to those who, after intensive study, are likely to advance knowledge; the fact, however, should be stated. Admittedly the chief function of a post-graduate teaching hospital is the provision of facilities that will enable practitioners to revise their knowledge and to keep themselves abreast of recent advances in medical science. But to study medicine in any of its aspects without a constant endeavour to discover something new is to be less than commonplace and to subserve ignoble ends. The directors of study at the Prince Henry Hospital will not be content with mediocrity, and for this reason, if for no other, both they and the members of the Post-Graduate Committee in Medicine will seek to keep strong the liaison already existing between the hospital and the University of Sydney. They will expect the university to make the same effort.

In addition to the fellowships in medicine and surgery, there has been founded what will be known as the Prince Henry Hospital Fellowship in Anæsthesia. It is intended that the appointee shall assist the lecturers in anæsthetics in the organization and control of the section of anæsthesia and in the instruction of post-graduate students. Preference will be given to those who intend to undertake anæsthesia as a specialty, and the laudable provision is added that candidates for the fellowship must be capable of undertaking research in the subject. The conditions of service will be the same as those attaching to the fellowships in medicine and surgery and the salary will be at the rate of £375 *per annum*.

The provision already made for post-graduate teaching at the Prince Henry Hospital is a matter for intense satisfaction. At the same time all concerned in its upkeep and control must recognize that little more than a start has been made. The fellowships already founded must before long be followed by fellowships in such special branches as pathology, ophthalmology and oto-rhino-laryngology. This is only one part of the work waiting to be done at the Prince Henry Hospital. The Board of Management is faced with many problems and many difficulties. No attempt need be made at present to describe them; but we would insist that the board and the Post-Graduate Committee must receive financial and other necessary assistance from the Minister of the Crown, who, having transformed the hospital into a post-graduate teaching institution, is ultimately responsible for its fabric.

Current Comment.

THE TREATMENT OF CARDIAC IRREGULARITIES.

George Fahr has made a valuable contribution to the methods of treating irregularities of the heart.[1] As auricular fibrillation constitutes almost half of the cases of cardiac irregularity, he deals with that condition first. He emphasizes the fact that auricular fibrillation exerts a very harmful effect upon the pumping mechanism of the heart. Thomas Lewis has shown that auricular fibrillation reduces the output of the heart and also may increase the venous pressure, even in an animal whose heart is otherwise normal. A very serious condition arises when auricular fibrillation supervenes upon a valvular defect or myocardial damage. Fahr reminds us that patients may do perfectly well for years with mitral stenosis, but cardiac failure follows the onset of auricular fibrillation. Dropsy and breathlessness may then ensue with a life of invalidism. In the human subject untreated auricular fibrillation is followed by an increase in venous pressure and diminution in the output of blood leaving the ventricles during each minute. The tendency for the blood pressure to fall, however, is usually counteracted by means of vasomotor adaptation. The reduction in ventricular output is due largely to the increased rate of the ventricles and to the many premature and abortive contractions of these chambers as well as auricular paralysis. When administered in sufficient quantity, digitalis will reduce the ventricular rate to the optimum. With the reduction in ventricular rate the number of abortive and premature contractions becomes less. The beneficial effect of digitalis in auricular fibrillation is due to its partially blocking conduction from the fibrillating auricles to the ventricles. It is known that digitalis enhances the mechanical efficiency of the cardiac muscular fibres; but when auricular fibrillation is present the reduction in the ventricular rate is as great a factor in effecting compensation in cardiac failure.

Fahr considers mainly the use of preparations of *Digitalis purpurea*, as these are now carefully standardized in terms of the cat unit. He mentions only briefly the preparations from *Digitalis lanata*. In Fahr's opinion one cat unit (0·65 cubic centimetre of tincture or 0·065 gramme of the United States Pharmacopœia powdered leaf) to each 4·5 kilograms of body weight, when anchored in the body, gives very nearly the optimal effect in lowering the ventricular rate in auricular fibrillation. This is generally a perfectly safe quantity. When this quantity does not effect the requisite diminution in ventricular rate, an additional 10% to 30% will certainly do so. Not more than one patient in fifty will exhibit evidence of digitalis intoxication with this amount, and then the toxic effects will not be serious. If the digitalis administration is suspended for 24 to 72 hours, the manifestations of intoxication will be eliminated. Fahr states that the anchored digitalis is removed from the body at the rate of about one cat unit daily. Doses of digitalis of this amount will not reduce the volume flow through the coronary arteries nor damage the myocardium. One cat unit daily may be given to a man of 68 kilograms (150 pounds) for years without any danger. When vomiting precludes oral administration, the same dose as is given by mouth may be administered by the rectum. Whether digitalis is given as the tincture or the powdered leaf Fahr considers to be immaterial. When the patient is only moderately decompensated, Fahr considers that seven days may be taken for optimal digitalization. In such a case 21 cat units

[1] *The Journal of the American Medical Association*, December 17, 1938.

would be given to a patient weighing 150 pounds over the period of seven days. Six cat units would supply the loss of digitalis in the six days following the day on which the drug was first given, and fifteen cat units would be required for the optimal amount to be anchored in the body. Many of Fahr's dispensary patients are not seen until the lapse of a week. When the patient returns and the pulse is over 70 while he is at rest and not excited, Fahr prescribes two cat units daily until the rate declines to between 60 and 70. When this rate is attained he gives one cat unit daily. In dispensary practice Fahr often administers the calculated optimal amount within a shorter period when the patient is badly decompensated. In his hospital practice he generally endeavours to obtain the optimal effect more quickly. As a rule he gives 15 cat units to the 150-pound patient within the first thirty-two hours. One-third of the calculated quantity is given at once and one-sixth every eight hours for four doses. An attempt is made to maintain the ventricular rate at the optimal point by the exhibition of one cat unit daily when the optimal rate has been reached at the end of from forty to forty-eight hours. When this rate has not been attained Fahr gives two cat units daily until the rate is between 60 and 70. Thereafter one cat unit daily will generally hold the rate at this point.

Fahr admits that there are some exceptions to his rules for the administration of digitalis. He observes that occasionally digitalis seems to have no effect on conduction of the impulse from the auricles to the ventricles, and accordingly does not slow the ventricular rate. A small proportion of patients exhibit toxic manifestations before the optimal results in reducing ventricular rate can be attained. Some patients require more than one cat unit to ten pounds of body weight anchored in the body in order to produce optimal slowing of the pulse rate. A very few patients require less than the calculated optimal dose. This is usually found in coronary disease or in other conditions that in themselves reduce conductivity through the bundle of His. In hyperthyreoidism, as is well known, digitalis usually fails to lower the ventricular rate, and Fahr correctly remarks that its value is questionable. He very pertinently states that in toxic conditions associated with fever digitalis dosage should be smaller. It is unwise in such conditions to go beyond one-half of the usual dose.

Fahr in his communication likewise deals exhaustively with the administration of quinidine and its dangers, and also with paroxysmal tachycardia and heart block. His dissertation is particularly valuable.

COMPARATIVE MEDICINE.

Man is indebted, in part at least, to veterinary science for many of the advantages in health that have been brought to him during the past hundred years. The veterinarian has advantages over the medical scientist in the study of disease; for he can destroy a subject when necessary and at any stage in the development of the morbid process, he can conduct *post mortem* examinations without formality, and he can experiment with almost unlimited freedom in therapeutic procedures. Probably these opportunities have not always been readily seized; but there is now ample evidence that the veterinary scientist is aware of the necessity of research and is conscious of his importance in the preservation of the health not only of man's flocks and herds, but also of man himself. In an address delivered before a meeting of the Section of Comparative Medicine of the Royal Society of Medicine, Thomas Dalling, the president of the section, discussed a number of recent advances in the knowledge of disease of domestic animals, all of which are of general interest and several of special interest to the medical practitioner.[1] One is the method of immunizing lambs against *Clostridium welchii*. This organism produces its pathogenic effects by a toxin elaborated in and absorbed from the intestine. As a measure of prophylaxis the pregnant ewe is injected with two doses of a formalinized culture of *Clostridium welchii*, the second dose being given a few days before parturition. The lamb is not born immune, but obtains the antibodies from the colostrum and is rendered immune within a few hours. The antibodies continue to be absorbed from the intestine into the general circulation until the lamb is four days old. This proven fact opens up possibilities in human immunization that have hitherto scarcely received consideration.

A second point of interest is brought out in Dalling's discussion of *Clostridium tetani*. A method of active immunization of horses by means of a tetanus toxoid has been evolved. "Horses so immunized will withstand natural infection probably for years." Dalling draws attention to the possible value of this procedure in the protection of soldiers against tetanus in time of war.

After dealing with the absorbingly interesting virus diseases and the diseases caused by mineral deficiencies, Dalling discusses lymphomatosis of fowls. In this disease aggregations of lymphoid cells appear in the tissues, particularly nervous tissue. Large tumour-like bodies may form. The disease itself or a susceptibility to it is transmitted in the egg, and it can be eliminated from a flock by continuous breeding from the survivors alone. Dalling feels that the cause, which is still unknown, may be closely linked up with the cause of tumour formation generally. The results of investigation of this condition might have an important bearing on the study of human disease.

Medical men as a whole do not pay sufficient attention to comparative medicine. Even such a cursory examination of the subject as is entailed in the perusal of Dalling's short paper will be of value.

[1] *Proceedings of the Royal Society of Medicine*, November, 1938.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

The Action of "Progesterone".

Bernhard Zondek and Samuel Rozin (*The Journal of Obstetrics and Gynæcology of the British Empire*, December, 1938) have investigated the effect of the intramuscular injection of "Progesterone" during the normal cycle of women, in the treatment of amenorrhœa and during pregnancy. Daily injections of 10 milligrammes of "Progesterone" administered to normally menstruating women for five days (usually the seventh to twelfth day of the cycle) caused bleeding of several days' duration after an interval of sixty hours (usually on the fourteenth day of the cycle). This hæmorrhage is a pseudo-menstruation. The normal cycle need not be disturbed by this event. Another bleeding can occur within the same cycle. During the "Progesterone" treatment the breasts were swollen and painful, and there was complaint of abdominal pain, all of which disappeared when bleeding commenced. Five days' treatment with daily injection of 10 milligrammes of "Progesterone" can produce hæmorrhage in secondary amenorrhœa without preliminary treatment with follicular hormone. In primary amenorrhœa, however, this mode of treatment fails. The reason perhaps is that in primary amenorrhœa the mucous membrane is atrophic, while in secondary amenorrhœa a certain amount of proliferation of the uterine mucosa is present. The authors conclude that "Progestérone" stimulates the anterior pituitary, causing increased secretion of the follicle-ripening hormone, which in its turn secondarily induces increased production of follicular hormone. In a few cases of secondary amenorrhœa they succeeded in initiating the action of the cycle by means of "Progesterone" therapy. They failed to initiate bleeding during pregnancy following administration of "Progesterone".

Treatment of Vaginal Moniliasis with Silver Picrate.

Herman A. Shelanski and Frank M. Kern (*American Journal of Obstetrics and Gynecology*, January, 1939) discuss the diagnosis and treatment of infections of the vagina with *Monilia albicans*. The organisms are oval in form, measuring from five to six microns in length and about four microns in breadth. The mycelia, on the other hand, vary both in thickness and in length, showing many intermediate stages between budding and a typical mycelium. The lesions produced are caseous white patches on the vaginal mucosa and cervix. There is usually a copious white discharge, acetic in odour, which is irritating to the neighbouring skin. In the severer cases an acute dermatitis results and is often associated with the formation of pustules. In the older chronic cases the vagina appears dry and encrusted. A vaginal smear from one of the caseous white areas is diluted with normal saline solution and a drop is examined microscopically under the low and then under the high power dry lens for yeast buds and mycelia. The diagnosis may be confirmed by culture on Sabouraud's agar for twenty-four hours. The infection will respond to treatment with a 2% aqueous solution of gentian violet. The authors preferred to cleanse and dry the vagina, and to paint it with a 1% aqueous solution of silver picrate. They then insufflated the vagina with five grammes of 1% silver picrate in kaolin. This treatment was repeated every week. The insufflation was omitted when the lesion had produced a dry vagina. The authors treated twenty-eight patients. After two treatments sixteen were symptom-free and both smears and cultures were free from organisms. Ten were cured after four treatments. The remaining two required five and eight treatments respectively.

Ovarian Transplantation.

Z. Üstün (*Monatsschrift für Geburtshülfe und Gynäkologie*, September, 1938) describes his experiments with the transplantation of ovarian tissue in guinea-pigs. In one group the ovary of a young animal was implanted in the back muscles of an adult guinea-pig; in the second series one ovary of an adult animal was removed and placed in the back muscles; in the third group both ovaries were removed and an ovary from a young animal was implanted in the back; in the fourth group, after removal of both ovaries, slices of an ovary from a young animal were implanted into the back and then 30 units of œstrin were injected daily for ten days. In no instance was there any evidence of normal activity in the transplanted ovarian tissue. Necrosis was observed, associated with extensive fatty infiltration in the graft. A similar result was observed when the ovary was embedded in the mesovarium or mesosalpinx.

Intraabdominal Hæmorrhage during Pregnancy.

G. Gaehtgens (*Monatsschrift für Geburtshülfe und Gynäkologie*, September, 1938) discusses the ætiology of intraabdominal hæmorrhage during pregnancy. He considers that it is mainly due to anomalies of placentation, and especially to *placenta accreta*. The details of such a case are given in which in a previous delivery a septic puerperium had been followed by a long period of amenorrhœa. The author divides the cases into three groups. In the first are those with pain and collapse followed by anæmia, which may be confused with accidental hæmorrhage. In the second group the symptoms are similar, but abate and then recur. In the third group there may be an absence of symptoms; delivery may be normal and no signs may develop until the third stage. The only treatment is hysterectomy followed by blood transfusion, but in many of the cases so much blood has been lost that recovery is impossible, as was noted in the author's case. In a few instances hæmorrhage may be due to rupture of vessels in the broad ligament.

Transitory Fever of the New-Born Infant.

W. Hagedorn (*Monatsschrift für Geburtshülfe und Gynäkologie*, September, 1938) discusses the ætiology of fever in the first days of life of the infant. In the majority of cases it is a thirst fever and is associated with considerable loss of weight. It appears to be more frequent with males than with females, though parity and the method of delivery play no part in its incidence. The patient's temperature reaches its maximum between the third and fifth days, when the loss of birth weight is maximal. The average temperature noted was from 37·6° to 38·5° C. (99·7° to 101·3° F.) and was seen generally when the loss of weight was from 500 to 600 grammes. The obvious prophylaxis is to administer abundant fluids during this period. The prognosis is good and no after-effects have been noted.

A Simple Method of Constructing a Vagina.

Lawrence R. Wharton (*Annals of Surgery*, May, 1938) presents a report of four cases in which a vagina has been constructed by a simple method. Other operative procedures previously devised include the use of skin grafts, of prepared tubular flaps, of the *labia minora* and of the pelvic peritoneum. A double loop of ileum has been used and also the sigmoid colon. The author's method embodies two new features. One is based on the principle that vaginal epithelium has remarkable powers of proliferation and in a relatively short time will cover a raw surface. The second new feature is the use of a plug or mould to keep the vaginal cavity open. The author conceived the idea that a large rubber condom, filled with some fairly firm substance, would provide a suitable mould for the future vagina. The operative technique is simple. An incision is made in the external mucous membrane or across the dome of the rudimentary vagina, and a space between the bladder and the rectum is created by blunt dissection. The plane of cleavage furnished by the fibro-areolar tissue between the layers of the subvesical and perirectal fascia is followed. There is little danger of perforation of either rectum or bladder if this layer is followed. The vaginal mould should be light and firm, and this is inserted into the space so created, and is maintained there for a

period of three weeks. During this period the patient is kept in bed. In his discussion of the results the author states that his patients are well satisfied and that coitus is satisfactory and painless. Portions of the vaginal wall removed up to two years after the operation show the histological characters of normal vaginal epithelium.

OBSTETRICS.

Climate in Relation to Eclampsia.

W. J. DIECKMANN (*American Journal of Obstetrics and Gynecology*, October, 1938) reviews the literature on the influence of weather and climate on eclampsia. Most of the writers seem to support the theory that eclampsia is more common in the cold, damp, unsettled weather. The author has noted that symptoms are usually intensified at any abrupt change in the temperature. The incidence ranges from 0 in some cities to 2·85% in Algiers, in Africa, and 7·2% in Charlotte, North Carolina. There is some evidence to support the view that a high average temperature, a small range temperature and a high measure of rainfall are factors. This is negatived to some extent when one examines the figures for India, where the incidence is 3·8% for Hindus and 1·66% for Mohammedans; the latter are meat-eating, whilst the Hindu eats no meat.

Cæsarean Section in Massachusetts.

R. L. DE NORMANDIE (*The New England Journal of Medicine*, December 1, 1938) publishes a report on the Cæsarean sections carried out in Massachusetts in 1937. The report concerns 2,106 abdominal deliveries, which, since the total births was 63,988, is an incidence of 1·0 in 30·3 births. The low operation was done 1,178 times and the death rate from all operations was 3·1%. In the author's opinion many of the indications were bizarre, and he expresses the opinion that many of these Cæsarean sections were entirely unnecessary; 788 of the operations were performed by surgeons. He has no objections to a well-trained surgeon doing a Cæsarean section, but he objects to a physician with no experience in operative obstetrics calling on a surgeon to get him out of his difficulties. In such a case the only procedure the surgeon knows how to carry out is a Cæsarean section, and he is only too glad to do it.

A Study of Seventy-Five Transfusions with Placental Blood.

B. S. GRODBERG AND E. L. CARY (*The New England Journal of Medicine*, September 29, 1938) record the study of seventy-five transfusions with placental blood. At the Boston City Hospital the writers have established a placental blood bank. The method is to allow the blood to flow from the promptly cut cord into a sterile flask containing anticoagulant. The blood is stored away and tested for its group and sterility, and the mother also is subjected to a test for syphilis. The average amount of blood collected from each patient was 105 cubic centimetres, with a variation from 35 to 215 cubic centimetres. It was found that the fœtal blood group varied from the maternal in 33% of the cases; in one case of triplets the mother's blood was group IV, the blood of two of the triplets (identical males) was group IV, and that of the third (a female) group II. The authors have demonstrated that babies have their own fixed blood agglutinogens at birth, and no change in group was noted before discharge from the hospital. The blood before use was warmed to body temperature and filtered through six to eight thicknesses of gauze. The gravity method was used, and as one sample of blood ran out the next was given with as little mixing as possible. The average amount used was 325 cubic centimetres up to 525 cubic centimetres. The average number of specimens of blood used was three, and the greatest number was five. The average time the blood was kept was six days, the longest thirty days. The reactions that occurred numbered 3%; which was lower than that occurring when fresh blood was used. It is held that placental blood is always available for emergency transfusions and in some instances is life-saving.

Mongolism.

E. KEHRER (*Monatsschrift für Geburtshülfe und Gynäkologie*, July, 1938) gives the details of four cases of mongolism in new-born infants and of a rare instance of familial mongolism. In general the diagnosis is easy, because of the more or less characteristic facies, generalized muscular atony, flaccidity of the subcutaneous tissues, especially of the skin of the neck, and shortening of the terminal phalanx of the fifth finger. While it is generally considered that such cases occur with *multiparæ* over forty years of age, this was noted in only one of the author's series. In one instance the mother had suffered from *encephalitis lethargica* two years previously, and in another close relationship between the parents may have played a part.

Relation between Blood Plasma Proteins and Toxæmias of Pregnancy.

E. F. DODGE AND T. T. FROST (*The Journal of the American Medical Association*, September 19, 1938) discuss in a preliminary report the diet of the antenatal patient and its relation to the serum albumin and serum globulin contents. It was noted that there was a lowering of the blood plasma proteins in normal pregnancy, which became more pronounced as toxæmias developed. The fall is in the nature of a fall of the albumin and a compensatory rise of the globulins. The authors have found that by increasing the protein intake of their patients during pregnancy they have been able to reduce the incidence of eclampsia among their patients to nil. Not only was the incidence of toxæmia lessened, but those patients showing mild toxæmia were greatly benefited by the addition of extra protein in the form of six eggs per day.

Hydrops Fœtalis (Erythroblastosis).

VICTOR BONNEY AND H. J. S. MORTON (*The Proceedings of the Royal Society of Medicine*, July, 1938) report the history of a patient who on three occasions failed to produce a healthy child. The first child became jaundiced soon after birth and died in forty-eight hours. The second was premature and stillborn. A *post mortem* examination was made on each of these children. A large liver and spleen, together with free bile-stained fluid in the abdominal cavity, led to the diagnosis of congenital syphilis. The blood of the parents failed, however, to give a positive reaction to the Wassermann test. When the patient became pregnant again it was decided to deliver the child by Cæsarean section before term, whilst the heart sounds were good. Unfortunately rapid changes set in and were accompanied by toxic symptoms. Examination of the fœtus revealed failure of the hæmatopoietic system associated with œdema of all organs. The pathological report was that the condition coincided with fourteen other cases of the œdematous form of *erythroblastosis fœtalis*. The diagnosis of this condition is important, as the *post mortem* findings are allied to those of congenital syphilis; even the changes in the bone ends are somewhat similar. In the past enlargement of the placenta was thought to be characteristic of congenital syphilis; but it would appear that it is really a characteristic of the œdematous form of erythroblastosis. In the event of a child being born alive, a certain number are saved by blood transfusions. Those with hydrops are usually stillborn. Thus *hydrops fœtalis*, *icterus gravis neonatorum* and *anæmia hæmolytica neonatorum* are all manifestations of the one condition, namely, failure of the hæmatopoietic system.

Unusual Cause of Hyperemesis in Late Pregnancy.

P. OEHLKE (*Monatsschrift für Geburtshülfe und Gynäkologie*, July, 1938) discusses the various causes of vomiting which occur in the later months of pregnancy, and gives the details of a case in which the vomiting was due to the presence of a tapeworm. Despite treatment the worm could not be dislodged, and signs of severe anæmia associated with mild pyelitis developed. Because of the onset of jaundice and coma, in spite of intensive glucose therapy, the pregnancy had to be terminated.

British Medical Association News.

SCIENTIFIC.

A meeting of the South Australian Branch of the British Medical Association was held at the University of Adelaide on November 24, 1938, Dr. P. T. S. Cherry, the President, in the chair.

Clinical Dietetics.

Dr. A. R. Southwood read a paper entitled "Clinical Dietetics" (see page 467).

Dr. R. G. Burnard expressed the appreciation and thanks of the members for Dr. Southwood's paper. The discussion of the physiology of dietetics as the basis for subsequent remarks made an interesting and helpful approach to the problems involved.

Dr. Southwood was asked to enlarge on his experience of the use of insulin in non-diabetic conditions, more particularly in wasting diseases. In regard to obesity, the cause appeared in some cases to be, apart from grosser endocrine disorders, excess of carbohydrate intake; but in many instances it was a matter of heredity. In these the problem was analogous to that of trying to make a race-horse out of a draught horse, and the danger was that by treatment one might spoil a good draught horse and produce a very indifferent racer. There seemed to be a need for more definitely determining the cause in an individual case of obesity. The treatment in most cases amounted to starvation, though sometimes thyreoid extract was helpful.

The ketogenic diet for the treatment of pyelitis had been useful, though now it was superseded by the sulphanilamides. The results with drugs of this type in acute pyelitis were sometimes quite dramatic.

The avoidance of red meats, soups, condiments, tea and coffee, alcohol and tobacco, in Dr. Burnard's experience, made patients suffering from hyperplesia much more comfortable.

In regard to the many dietetic cults and fads of which Dr. Southwood spoke, many of these were literally being ridden to death. A sound application of the materials and methods of clinical dietetics as presented by Dr. Southwood should prove the most satisfactory method of countering the ill effects of irregular practitioners in this respect.

Dr. Eugene McLaughlin said that a state of simple obesity could not be maintained on a diet of low caloric value. The intake of alcohol was often forgotten or concealed by the patient.

It was necessary for a medical practitioner when prescribing a diet for the obese, to provide one which was reasonably varied and attractive; otherwise the course prescribed would not be observed. It was important to provide a well-balanced diet and to see that the meals were correctly spaced over the day. The first weeks of reduction were best carried out under nursing supervision, and the loss of one pound a week should be regarded as sufficient.

In reply, Dr. Southwood stated that he had anticipated a more argumentative discussion on some of the points he raised. He thought, for instance that the suggested relationship between imperial greatness and the intake of beef and beer might have provoked opposition.

With the details raised by Dr. Burnard and Dr. McLaughlin, concerning the treatment of obesity, he was in general agreement.

Dr. Southwood did not consider that moderate indulgence in alcohol and tobacco was especially harmful to the ordinary subjects of high blood pressure.

Dr. Southwood thanked the various speakers for their remarks and considered that they had dealt rather leniently with him.

A meeting of the New South Wales Branch of the British Medical Association was held at Saint Vincent's Hospital, Darlinghurst, on October 20, 1938. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Lung Abscess.

Dr. R. J. Taylor showed a female patient, aged twenty-eight years, who had been admitted to hospital on July 9, 1938, and discharged on August 30, 1938.

For years the patient had suffered from asthma. In May, 1938, she had a tooth removed and difficulty was experienced in its removal. Afterwards the socket discharged pus and was not completely healed at the end of six weeks. Six weeks after the extraction of the tooth the patient had a severe attack of asthma, which lasted for three days. Later she had a severe cough with sputum. Five weeks before the time of the meeting she coughed up a large amount of blood. Three days later she commenced to cough up purulent sputum, which, she said, had a foul taste. She lost weight.

On examination at the time of her admission to hospital it was noted that the patient, a young woman, was disturbed by paroxysmal coughing. The movements of the upper part of the left side of the thorax were diminished. The percussion note was impaired at the left apex anteriorly and posteriorly. Vocal resonance and fremitus were normal. The breath sounds were vesicular, but fainter at the left apex, and were accompanied by scattered rhonchi. Examination of other systems revealed no abnormality. While the patient was in hospital she had a swinging temperature, varying between 37·2° and 38·9° C. (99° and 102° F.). The temperature fell to normal on August 10, 1938. Postural treatment was carried out.

On July 11, 1938, X ray examination revealed in the left axillary region a large cavity, half filled with fluid. The appearance was that of a tuberculous cavity on July 15, when it was noted that the right lung appeared clear.

On July 26, 1938, the cavity had increased slightly in size and still contained a large collection of fluid; a further increase in size was noted on August 6.

On August 12, 1938, "quite an extraordinary improvement" had taken place and the cavity was reduced to less than half the size it had previously been. Further diminution in size was noted on August 20. The sputum was examined microscopically, but no tubercle bacilli or hooklets or scolices were found. A guinea-pig after inoculation developed tuberculosis. Further examination of the sputum revealed abundant staphylococci in films and a few pneumococci. On culture an abundant growth of *Staphylococcus aureus* was obtained; the growth of pneumococci was scanty. A neutral leucocytosis (22,000 cells per cubic millimetre) was present with a moderate shift to the left. The erythrocytes numbered 2,800,000 per cubic millimetre, the hæmoglobin value was 53%, and the colour index 0·94. The Casoni test yielded no reaction.

Von Recklinghausen's Disease.

Dr. Bruce Hall showed a man, aged sixty-four years, who was suffering from von Recklinghausen's disease. The patient had presented himself at hospital complaining of pain in the left renal region of six weeks' duration. X ray examination revealed a large calculus in the lower pole of the left kidney and extensive changes characteristic radiologically of von Recklinghausen's disease. These changes were present in the lower part of the vertebral column and in the pelvis. Further radiological investigation at a later date revealed similar changes of varying degree in other parts of the skeleton, including the skull. There was also evidence of unusual calcium deposition in arteries and in the lungs. Vascular hypertension was present. Biochemical investigations revealed a serum calcium content of 12·5 milligrammes and a serum phos-

phorus content of 11·0 milligrammes *per centum*. The blood urea content was 41·0 milligrammes *per centum*. By the urea concentration test it was found that the patient concentrated urea to over 2% in two hours. Dr. Hall stated that other investigations had been carried out, but that they were not relevant or had given normal results.

In discussing the case Dr. Hall said that the chief points for comment were the raised blood phosphorus level and the signs of fairly active calcium deposition in unusual situations. The raised blood phosphorus level, although uncommon, was a well-recognized finding and was apparently related to metastatic calcium deposition. Cameron had stated that calcium could be slowly deposited from the kidney tubules into the parenchyma, the concretions eventually leading to inflammatory changes, sclerosis and contracted kidney. Cameron held that in such cases the blood phosphorus might not be below normal, but might even be increased. Dr. Hall went on to explain that the patient had only very recently presented himself at hospital and the condition needed further elucidation in some respects. In spite of the results of the tests relating to renal function, however, Dr. Hall suspected that further observation would probably place the case in the clinical subgroup (described by Allright and others) in which renal changes led to interference with phosphorus excretion by the kidney and elevation of the blood phosphorus level.

Hæmochromatosis.

Dr. Hall also showed a woman, aged forty-seven years, who exhibited bronzing all over and histological skin changes in keeping with a diagnosis of hæmochromatosis. The patient also had hyperglycæmia and heavy glycosuria. As far as the diabetic condition was concerned, she showed a very poor response to insulin therapy, which appeared to be in keeping with the inability of a damaged liver to store glycogen. The patient had undergone cholecystectomy for gall-stones three years previously.

Parathyreoid Adenoma Associated Primarily with Urinary Lithiasis.

DR. DOUGLAS MILLER showed a male patient, aged twenty-one years, who for ten years had formed calculi in both kidneys. The calculi had been removed from the kidneys and ureters by different routes on various occasions. The patient was constantly suffering from renal colic and often passed calculi which were sometimes of a surprising size. He had no complaints referable to the skeletal system. X ray examination revealed calculi in both kidneys; slight decalcification was present in the long bones, and there was a suggestion of early cyst formation. The blood calcium content on two examinations was found to be 16·0 and 14·0 milligrammes *per centum*. Surgical operation was performed in March, 1938, and a parathyreoid adenoma was removed from behind the left retrothyreoid fascia. The adenoma measured 2·5 by 2·0 centimetres. After the operation the patient had latent tetany for a few weeks, but this was readily controlled by small doses of parathormone. The post-operative blood calcium content on four occasions was 11·0, 11·0, 9·0 and 6·0 milligrammes *per centum*. The last estimation was made in August. X ray examination made in August showed that the calculi had disappeared from the left kidney and that those in the right kidney were much reduced in size. The bones were greatly increased in density.

Hyperadrenalism: Male Pseudo-Hermaphroditism.

Dr. Miller also reported a case of hyperadrenalism. The patient, who was a male pseudo-hermaphrodite, had at birth been regarded as a boy with hypospadias. At the age of four years he had been operated on for hypospadias. During the first year of life he did not grow at all. At two and a half years of age he started to grow rapidly, and when he was four years of age he was over four feet in height. His external genitals were well developed and he had pubic hair. He was over five feet high at the age of six, and did not grow since then. He had had to shave his face from the age of seven. He was very studious and fond of books. He was attracted to adult women, and was very jealous of people coming between him and the object of his affections. He was apt to have an uncontrollable temper over such things, and could never be allowed to leave home.

On examination he was found to have a dark skin and heavy axillary and pubic hair. He had a strong muscular and masculine body. His voice was deep and masculine. His interests were those of a youth of eighteen.

At operation no right adrenal gland was found; the left adrenal was four times the normal size. The genital organs were those of a female. No male gonads were present. A vagina opened into the urethra, which opened on the perineum. While the patient was under an anæsthetic the systolic blood pressure rose to 170 millimetres of mercury. The patient died after operation. The specimens were female organs. The suprarenal gland showed great increase in cortex, and in the androgenic layer pronounced fuchsinophile preponderance was present.

Chromophobe Adenoma of the Pituitary Body.

Dr. Miller showed a series of patients who were suffering from chromophobe adenoma of the pituitary body.

A female patient, aged fifty-six years, had been treated for three years with deep X ray therapy. She had gone blind in the right eye, had a temporal hemianopia, and could only count fingers with the left eye. She was operated on by the transfrontal route, and a cystic tumour was evacuated from the pituitary. She made a very rapid recovery of vision in the left eye, acuity coming to $^6/_{12}$ in a week. Some months later her visual acuity again became impaired. Further deep X ray therapy effected an improvement and the visual acuity had been kept at $^6/_{12}$ for one year.

A man, aged fifty-two years, had suffered from progressive loss of vision for nine months. He was found to have a bitemporal hemianopia with ability only to count fingers in one eye and visual acuity of $^6/_{36}$ in the other eye. After two weeks of deep X ray therapy the field of vision in one eye had commenced to open out on the nasal side, and visual acuity had improved to $^6/_{24}$. It was proposed to continue with X ray treatment for the present.

A female patient, aged thirty-four years, had noticed trouble with vision for some months. She had suffered from severe retroorbital headache. In July, 1938, the fields of vision had shown only slight depression of the upper temporal quadrant on both sides. One month later there was a bitemporal quadrantic hemianopia on both sides. As the acuity had not suffered it was decided to give deep X rays. After the first week of this treatment the patient had intense headache and became practically blind in the left eye, developing complete hemianopia in the right. Early transfrontal operation was performed and a tense degenerate tumour was dealt with. The patient's vision made a rapid recovery in both eyes.

A Septic Thumb.

DR. V. M. COPPLESON showed a woman, aged fifty-seven years, who on May 15, 1936, developed an inflammation of the left thumb. One week later she showed it to a doctor, who told her that she had a germ under the nail; the doctor opened it slightly. A fortnight later it was opened further under the nail, and a month later the nail was removed. The condition did not improve and was very painful. The thumb was again opened at a later date and the "bone was scraped". On December 17, 1936, the thumb was removed at the terminal phalanx; but this had no effect on the condition and the pain was still severe. In February and April, 1937, she attended a hospital, where she was advised to have the thumb taken off at the metacarpo-phalangeal joint. In the meantime she was treated by hot air and the condition became very much worse. She saw another doctor in May, 1937, who removed a further portion of the thumb, still without any effect at all on the pain.

When first seen by Dr. Coppleson, on June 16, 1938, she stated that "all the time it felt as if it was full of quicksand, twisting, burning and throbbing". She was sleeping very little at night and had pain when she moved

the thumb in any way. She could not even bear to touch it and could not bear anything around the tip. The patient kept her thumb bound up in the palm of her hand so that nothing could touch the end of the amputation stump, which was exquisitely tender. An injection of "A.B.A." was given into the median nerve, with immediate relief. She returned a week later with pain still present.

On July 7, 1938, she was admitted to the Saint Vincent's General Hospital, where an operation was performed on August 4, 1938. About one inch of the radial artery was decorticated, and the metacarpo-phalangeal joint of the thumb, which was stiff, was moved under the anæsthetic. On August 18, 1938, the patient was discharged from hospital with complete relief.

Hypertrophy of the Breasts.

Dr. Coppleson's second patient was a married woman, aged twenty-eight years, who had complained of increase in size of both breasts during the previous four years. The increase had been more pronounced during the previous twelve months. Latterly dull aching pain had been present in both breasts. The patient had had no previous illnesses. She had two children, one aged twelve years and one aged nine years; she had also had one miscarriage seven years previously.

On examination both breasts were found to be greatly enlarged; they were extremely heavy. The breast tissue felt normal. On July 6, 1938, a plastic operation was performed on both breasts. An incision was made around the areola and downwards, curving outwards below; the flaps were dissected free. By an incision from the top of the breast lateral to the nipple and extending to the medial side below the nipple three-quarters of the breast tissue was removed. The remaining breast tissue was brought round and sutured on the lateral aspect. All redundant skin was removed and the wound was sutured. The other breast was similarly treated. Both breasts were examined and the sites for the nipples was chosen. A circular piece of skin was excised at the selected spot and the nipples were brought through and sutured to the skin. The result was entirely satisfactory.

Actinomycosis of the Jaw.

Dr. Coppleson also showed a woman, aged twenty-five years, who had had an impacted wisdom tooth removed from the right side of the lower jaw five months prior to her being seen by Dr. Coppleson; an abscess developed at the site and also three swellings on the external aspect of the lower jaw on the same side. The abscess inside the mouth discharged for two months. The swellings outside the jaw remained painful and increased slightly in size until one month prior to the patient's admission to hospital, when they were incised and yellowish pus was evacuated. On admission the patient had a hard lump over the right angle of the jaw, which extended anteriorly in front of the masseter and downwards to the cornu of the hyoid bone. The patient was given potassium iodide and deep X radiation.

On examination of the discharge a streptothrix, often beaded and forming small clumps, was discovered in the thin pus from the sinuses.

X ray examination of the jaw revealed a fairly large area of bone erosion deep to the site of extraction of the lower right first molar tooth and behind the remaining molar. There was no evidence of sequestrum. Sepsis was present on the left side of the mandible and bilateral cervical ribs were present. X ray treatment had been commenced on May 31, 1938. This had been continued until October 11, 1938, when there was no discharge externally, although some came from inside her mouth.

Dr. Coppleson also showed a man, aged forty-nine years, who was first seen on June 26, 1938, complaining of swelling on the right side of the tongue of six weeks' duration. The swelling had been the same size for the whole time. No salivation, bad odour or discharge was present. The patient felt well; he had not lost weight.

On examination the right side of the tongue, as far as the mid-line, was hard and phlegmonous. The tongue was fixed on the right side, and on protrusion the tongue deviated to the right. The floor of the mouth was free and no ulceration or bad odour was present. The posterior surface of the tongue was covered by a grey leucoplakia. The right submaxillary gland was enlarged. The patient's speech was thick. He was given potassium iodide. The Wassermann and the Kahn tests elicited no reaction. The condition was regarded as one of parenchymatous glossitis.

On August 4, 1938, a woody phlegmon was still present, and the condition was then considered to be actinomycosis. The patient was seen in consultation by Dr. H. R. G. Poate, who suggested that some "Proseptasine" should be tried. He was accordingly given a course of this drug. On September 19, 1938, the condition was very similar, although slightly improved, and the patient was referred to Dr. H. Ham for deep X ray therapy. This had been given, with the result that the swelling appeared less, but the tongue was still shrivelled and fixed. The lesion was examined by Dr. Gunn, of the Veterinary School, who agreed that the condition was consistent with actinomycosis of the tongue as seen in cattle.

Lymphosarcoma.

Dr. Coppleson's next patient was a woman, aged eighty-two years, who had been admitted to Saint Vincent's Hospital on November 29, 1937, on account of a lump which had been present in the left axilla for six weeks. There was no history of injury. There was no pain, but tenderness was present on pressure. The lump had gradually increased in size. On examination the mass was hard and one inch in diameter; it was situated in the posterior axillary fold. It was firm, somewhat nodular in outline and tender to pressure, also freely movable. On December 1, 1937, the mass was removed under local anæsthesia. The patient was discharged on December 7, 1937.

Dr. A. H. Tebbutt reported that the tumour appeared to be several lymph glands now fused together in one enlargement. On bisection a succulent, greyish, rather homogeneous tissue was revealed. Outside the tumour was fatty tissue. Microscopic examination, in Dr. Tebbutt's opinion, showed the tumour to be a lymphosarcoma; he gave the following reasons:

1. An almost complete loss of lymph gland structure, the normal germ centres and lymph sinuses, and trabeculæ are not seen.
2. The predominant cells are round cells, with rather less deeply stained and somewhat larger nuclei than the small round-cell type, though these are also present in groups and strands.
3. There is a monotony and homogeneity of this round-cell structure, with little evidence of reticulo-endothelial proliferation or fibrous tissue production, and no Read Steinberg cells.
4. Above all there is an infiltrating of the capsule and the adjacent fat, with, in places, no capsule, but just a merging into fat.

Spindle-Cell Sarcoma.

Dr. Coppleson also showed a woman, aged thirty-seven years, who had first been brought to the casualty department by ambulance on account of a hæmorrhaging mass near her left shoulder. Inquiries revealed that the patient had had a small tumour removed from her left shoulder region ten years previously. This reappeared twelve months before she attended the casualty department on January 27, 1938. It was first the size of a walnut and had gradually increased in size until it measured approximately 20·0 centimetres (eight inches) in diameter. Latterly there had been some small hæmorrhages, which were easily caused by pressure. On the morning of her attendance at hospital there was a very severe hæmorrhage, which caused the patient to collapse. She was a Christian Scientist and refused all medical attention until the hæmorrhage became severe.

On examination the patient was extremely exsanguinated and bleeding was taking place from the large fungating

mass occupying the left subclavicular region. The mass was lobulated, covered with distended veins and was bleeding from its lower pole. On January 4, 1938, the tumour was removed, an extensive bare area being left. On February 17, 1938, a skin grafting was performed. A second and a third skin grafting were subsequently performed.

On August 10, 1938, the patient returned to hospital complaining of a mass on the left side of her neck, present for one month and increasing in size. On examination there was a small solid tumour about the size of a walnut, which was removed on August 18, 1938, two nodules being found to be present. The patient was then referred to the deep X ray therapy department for radiation.

A pathological report was made on February 3, 1938, by Dr. A. H. Tebbutt. He described a very large fungating tumour of the subcutaneous tissue of the chest wall. It did not show any evidence of mammary tissue. It weighed 2·25 kilograms (five pounds). Its cut surface showed a rather soft greyish tissue, in which were yellowish and opaque white necrotic areas. It had large thin-walled veins running through it. At first sight Dr. Tebbutt thought that it might be a Brodie's tumour, but it was not really cystic, and microscopically it contained no gland elements and apparently did not arise in the breast. It was a spindle-cell sarcoma, obviously rapidly growing, liable to necrosis and with large thin-walled vessels.

Dr. Tebbutt reported on August 25, 1938, that the two nodules were of similar tissue, with greyish cut surface. Microscopic examination revealed the same structure, that of a spindle-cell sarcoma (with the suggestion again that such a tumour might belong to the group of Schwannomata).

Carcinoma of the Ileum.

DR. W. MAXWELL showed a female patient, aged forty-eight years, who had been admitted to hospital complaining of constipation and black motions for three months with attacks of vomiting for the immediately preceding two weeks. She had lost 18·9 kilograms (three stone) in weight in the previous five months. On palpation of the abdomen a ladder pattern appeared, which was accompanied by a cramping colic. This was the typical appearance of small bowel obstruction of the gradual chronic type.

At operation a ring carcinoma was found 10·0 centimetres (four inches) proximal to the ileo-caecal junction, with almost complete occlusion of the gut and the usual obstructive changes in the bowel above. There were no enlarged glands and no secondary masses were present in the liver. An anastomosis was established between the small bowel 20·0 centimetres (eight inches) above the neoplasm and the transverse colon at the junction of its right and middle thirds.

The operation for resection was performed two weeks later, the whole bowel between the points of anastomosis being removed flush with the latter, together with the mesentery of the terminal part of the ileum, the ileo-caecal glands, and the glands up to the origins of the right colic and right branch of the middle colic arteries respectively. Pathological examination confirmed the diagnosis of carcinoma.

Carcinoma of Caecum with Abscess.

Dr. Maxwell then showed a patient, who on April 5, 1938, had an operation elsewhere for an inflammatory mass in the right iliac fossa which had given him "soreness" for four days; drainage had been instituted for what was considered to be a large appendiceal abscess. Four weeks later the abdomen was again opened for removal of the appendix, when an indurated mass was found posterior to the fundus of the caecum. On being consulted Dr. Maxwell thought the mass to be possibly a residual phlegmon, especially as the appendix was not visible and was thought to be involved in the mass, which was quite small. But on separation of the mass the tissue comprising it proved friable and the caecum was quickly entered, when a fungating mass became evident on the mucosal aspect. A tube was introduced into the opening and the hole in the caecum was approximated around it; the tube was further isolated by sutures drawing together the more mobile parts of the caecum and a mobile parietal peritoneum laterally.

Biopsy of a fragment showed the mass to be an adenocarcinoma, so three weeks later the abdomen was again opened by an oblique incision extending around from the loin and surrounding the fistulous opening left by the tube. The caecum, ascending colon *et cetera* were mobilized in the manner usually adopted with carcinoma of the caecum, and resection of the bowel and glandular drainage were carried out. The wound healed by first intention.

Dr. Maxwell also showed a man, aged fifty-three years, who on admission to hospital had complained of colicky pains in the abdomen for six weeks. Previous to this he had had treatment for a month for flatulent dyspepsia. He had lost 4·5 kilograms (ten pounds) in weight. On examination he was a spare individual, of muddy complexion and anæmic. On examination of the abdomen a ladder pattern appeared, which was accompanied by the usual colic. The patient was admitted to hospital for urgent operation, at which a ring carcinoma was found 27·5 centimetres (twelve inches) proximal to the ileo-caecal junction, with the bowel above distended to a diameter of about 12·5 centimetres (five inches). A lateral anastomosis was made between the proximal and distal loops 15·0 centimetres (six inches) on either side of the lesion.

At a secondary operation eighteen days later the loop, including the anastomosis, was resected, together with the mesentery and the contained regional glands. One of the latter had enlarged considerably since the primary operation.

Biopsy confirmed the diagnosis of adenocarcinoma.

Trigeminal Avulsion.

Dr. Maxwell's last patient was a woman, aged forty-two years, who had had periodic attacks of paroxysmal pain over the distribution of the first and second divisions of the right trigeminal nerve at intervals for two years. The attacks had rapidly become more frequent and more prolonged. Immediately before admission to hospital she had had an attack which lasted for three weeks; during this time opiates gave little sleep, and she refrained from taking any solid food because of the exacerbation of the pain caused by pressure upon the mucosa of the palate. A paroxysm could be readily induced by pressure over the upper alveolus and also over a small area below the outer canthus. Wind also brought on exacerbation. She had lost 6·75 kilograms (fifteen pounds) in weight in the two weeks before operation. All the upper teeth on both sides had been extracted. The patient had a typical *tic douloureux* involving the ophthalmic and maxillary divisions of the fifth nerve.

Operation was performed by the usual subtemporal approach, the trigeminal dural tunnel was opened immediately behind the ganglion, the nerve trunk was severed and a length of 18·0 millimetres (three-quarters of an inch) of the trunk was avulsed. The lids were sutured.

Anæsthesia and relief were complete, and the eye was reopened after six months. There was no sign of keratitis, nor was there any evidence of wasting of masticatory muscles. Dr. Maxwell said that he had observed this absence of wasting in five cases in which the motor as well as the sensory root was avulsed.

Tumour in the Neck.

DR. V. J. KINSELLA showed a male patient, aged fifty years, who had had a swelling in the left submandibular region for five years. This was thought to be the result of a chronic lymphadenitis of non-specific type. Tonsils and teeth were grossly infected and had recently been removed. X ray examination of the chest revealed quiescent pulmonary tuberculosis. The size of the swelling was thought to exclude a tuberculous lymphadenitis, and the position excluded branchial cyst.

Intermittent Obstruction of the Parotid Duct.

Dr. Kinsella also showed a male patient, aged forty-five years, who gave the typical history of intermittent parotid obstruction. A sialogram prepared by the use of "Neo-Hydriol" revealed an obstruction of the parotid duct, within the gland, one large branch supplying the lower part of the gland escaping. The possible causes of this obstruction and the available methods of treatment were discussed.

Carcinoma of the Cervix Uteri.

DAME CONSTANCE D'ARCY, DR. C. F. DE MONCHAUX AND DR. NELL FARRAR showed six patients suffering from carcinoma of the *cervix uteri* who had been treated by radiotherapy alone. All diagnoses had been confirmed by microscopy, a section of the cervical growth being taken at the time the radium was inserted. In five of the six cases the initial radium treatment (or radon treatment in one instance) had been followed by deep X ray therapy to the pelvis and both ilio-pelvic regions. It was considered that this procedure was most important for the complete radiotherapeutic treatment of carcinoma of the *cervix uteri*; it was preferred as a routine measure that at least two full courses of X radiation should be given, the first being begun as a rule about six to eight weeks after the initial radium treatment. Sometimes, depending on the local condition of the patient, a third course of deep X ray therapy was administered, all patients being kept under close observation at the follow-up clinic. The sixth patient shown had quite recently come under observation and the usual amount of radium had been inserted on August 30, 1938. In this case the immediate effect of radium on the primary lesion could be seen, and this patient would have X ray therapy in due course. Incidentally, on microscopy, the carcinoma in this instance was mostly of the transitional (uncommon) variety, being only in places typically squamous-celled. In the other five cases the growth was of the ordinary squamous-celled type.

In five of the cases radium had been used primarily, the average dose at Saint Vincent's Hospital being a total of 7,200 milligramme-hours, administered as one treatment. As a rule, 30 milligrammes of radium element were inserted into the uterine cavity (ensheathed in a rubber tube) and 20 milligrammes of radium were applied, in cork applicators, to the vaginal aspect of the cervix. This average quantity of 50 milligrammes of radium was left in for a period of six days (144 hours), an average total dose of 7,200 milligramme-hours thus being given. The screenage employed in these cases was two millimetres of platinum or its equivalent. In one of the cases radon was used primarily instead of radium, without any apparent difference in the quite satisfactory clinical result. A dose of 13,824 millicurie-hours of radon was used and the patient had two full courses of X ray therapy subsequently.

In two of the cases there was an interesting associated syphilitic condition, both patients having given positive Wassermann reactions when seen first at the clinic, prior to radium treatment. The malignant condition of the cervix was apparently superimposed on a primary specific lesion (a chancre); and another interesting feature of these two cases was that neither had shown the usual response of the primary neoplastic lesion to radium until the underlying syphilitic element had been treated by the usual antispecific means. In other words, the healing of the carcinoma was delayed by the associated specific disease, the primary growth beginning to respond to the radium treatment only after the institution of antispecific measures. Undoubtedly, it was stated, the primary syphilitic lesion in these two cases had something to do (as it so often had in cases of lingual carcinoma) with the induction of an ordinary squamous-celled epithelioma of the cervix. A Wassermann test should be carried out as a routine measure in all cases of carcinoma of the cervix among patients presenting themselves at any gynæcological department or radiotherapeutic clinic.

The present clinical condition of all the patients shown was quite satisfactory, with no evidence of residual or recurrent carcinoma and no apparent extension to the vagina, the parametrial tissues, the regional lymph glands, or to distant parts of the body. The period of time from the initial radium (or radon) treatment to the present demonstration varied from three and a half years (March, 1935) to seven weeks, and the age of the patients shown from thirty-three to fifty-eight years.

A MEETING of the Victorian Branch of the British Medical Association was held at Ballarat on December 3, 1938, DR. J. P. MAJOR, the President, in the chair. A report of part of this meeting was published in the issue of March 11, 1939.

Some Hitherto Unemphasized Aspects of Digestion.

PROFESSOR W. A. OSBORNE delivered an address entitled "Some Hitherto Unemphasized Aspects of Digestion". He said that in speaking on the subject of digestion he was following a well-beaten track. Until modern times, what with sialogues in the mouth and clysters in the rectum, the digestive system was attacked by about 80% of all the drugs in common use. A jest had been made by a Jewish doctor in the Middle Ages, who said that his Christian colleagues resembled their own Saint Peter in that they had "powers to bind and to loose"; and that was the greatest part of medicine in those days. In more serious vein Professor Osborne pointed out that there were many openings for future research on unsolved problems of digestion. Every attempt to establish the fact of hormonal direction of salivary secretion had failed, yet pytalism of pregnancy now seemed to have a hormonal basis. It was a humiliating fact that one of the oldest and important diagnostic signs—the furred tongue—had no explanation; it was not yet understood why a little upset in the colon should cause furred tongue or excitement of the conjunctival glands. It was recognized as a generalization that any level in the alimentary canal was influenced by what was happening in the level above and the level below, and, just as Mackenzie had made them look on the circulatory system as a whole, they must look on the alimentary canal as a whole when attempting to solve what might appear superficially to be a problem of one or other part of the digestive system.

Professor Osborne said that he was one of those who strongly objected to the proposition that if all the vitamins in the diet were right the teeth would be perfect. They had to face a biological fact that all the organs were subject to degeneration and that only the merciless operation of natural selection had caused survival. The snake wriggled because it had lost its limbs, and eyeless fish were found only in dark caves. When the teeth were bad nutrition was poor, and the girl with bad teeth was sexually unattractive and undesirable; but in modern times with dental art nutrition was good and the girl with defective teeth might be quite winsome.

What was the reason for the selective action of ptyalin in saliva on boiled starch. Dr. Ivan Maxwell had discovered that boiled or colloidal starch had a marked inhibiting influence on peptic action and that when starch was carried to the dextrin stage inhibition disappeared. Speaking of the stomach, Professor Osborne asked his audience to remember the changes in the histology of the mucous linings; the œsophagus was lined with many layers of stratified epithelium resistant to physical and chemical attacks, but the stomach had a single layer of delicate columnar epithelium exquisitely sensitive to osmotic changes; if the osmotic pressure was lowered those cells would swell and become water-logged; if the osmotic pressure rose they would shrink and separate. One of the major stomach functions was to render food iso-osmotic, to dilute food that was hyperosmotic, and to add salts if necessary to render it iso-osmotic. Dr. Apperly, at Richmond, Virginia, had contributed much towards the establishment and wider recognition of those facts. Another of his fellow workers, Dr. Leon Jona, had proved that salting the food to taste was in reality salting it to be precisely iso-osmotic with the blood and with the

stomach cells. Professor Osborne stated next that the capacity of the stomach to equilibrate the contents with the osmotic state of the walls was not an established function of the stomach before the age of six months, and that Nature had decreed that milk which was iso-osmotic should be all that entered it from birth to the age of six months. That principle had been forgotten in every single artificial feeding formula. Raw milk was correct physically; doped or watered milk might be correct chemically, but it was wrong physically. The late Sir Truby King had failed to recognize those facts. Milk was ordained to pass without cooling or exposure to the air into the stomach without contamination; yet babies were liable to get milk collected anywhere, treated with peroxide of hydrogen, doped, and altered in all sorts of ways, and it was not what Nature had ordained.

Professor Osborne, after stating that it was commonly said that the stomach digested part of its contents, expressed the paradoxical view that the stomach was not a digestive organ if digestion implied breakdown into products fit for absorption. The job of the stomach was to liquefy the food, to turn the protein of the food, which was in a solid state, into liquid peptones and albumoses, and to liberate fat from its envelope. Beaumont, around 1822, had contributed a great deal to knowledge of the functions of the stomach. He was a remarkable man, and practically everything he had stated had proved to be correct, though often challenged; he had missed only the "psychic flow" discovered by Pawlow. The stomach was a twofold organ; a transverse band divided it into an area with stormy peristalsis, which might be termed the "pyloric mill", and a cardiac area where the peristaltic ripples were delicate and hard to see. The significance was that digestion was a surface phenomenon; if the food had a solid consistency it was digested more satisfactorily. The surface of the stomach poured its secretions onto the surface of the food; that was the teleology of milk solidifying by rennet action; the solidification was an absolute necessity in the arrangement for surface digestion. The organ retreated on it, keeping up its grip. Galen had devoted half a chapter to the subject.

Professor Osborne then remarked that the "air space" in the stomach needed a lot more research before it could be properly understood. The air space in a healthy stomach remained fairly constant in its dimensions; if through contractions or alterations in posture the gas came in contact with the cardiac orifice, a remarkably quick reflex occurred, consisting of a rapid opening letting the air out. Another point deserving of a great deal of research was the thick glairy coating of the stomach noted by Beaumont and habitually reported on in modern times by the users of gastroscopes.

Professor Osborne said that Beaumont had described that even in mild gastritis the subject experienced great lassitude and distress. The way in which the physical prostration was produced was not yet understood; if the disturbance was a more general one it might be ascribed to vasodilatation, but that explanation could not be accepted for the lassitude in mild gastritis. He had had subjective experience of the phenomenon and was satisfied that the accompanying lassitude and weakness of the limbs was not due to deviation of the blood supply into the splanchnic area.

Professor Osborne went on to discuss what he had named "the metabolic flare". He linked together three things: (*a*) the opening of the ileo-caecal sphincter when food entered the stomach, (*b*) the excitement of the colon, and (*c*) the elevation in metabolism as soon as food entered the stomach. The relief experienced when one drank a cup of tea or nibbled a biscuit suggested that a "hold-up" in the alimentary canal was a "hold-up" at the ileo-caecal sphincter. The metabolic rate rose at once when food entered the stomach and could not be due to the specific dynamic action of certain amino-acids. Whatever the explanation, within one or two minutes there was a rise in the metabolic rate. In a state of starvation, such as a savage, for example, was liable to have to withstand, the colon was not active, the ileo-caecal valve was in a state of spasmodic clonus, and the metabolism was low. Clive had referred to the action of his Indian troops, who had said that when rations were scarce they would drink the water in which the rice was boiled and the British troops could eat the rice. The natives knew that they could withstand starvation better than the other troops; they did it by the simple process of lowering metabolism. Professor Baldwin Spencer and others had observed that Australian aborigines would fall asleep with hoar frost on their skins. Professor Osborne commented that on the entry of food into the stomach an "all clear" signal went down, the ileo-caecal valve opened, the colon became active, and the metabolic flare occurred and thus allowed the utilization of the food by the body.

He then referred to the very puzzling problem of the vomiting which occurred in such conditions as pregnancy, acute glaucoma and cerebral tumour, and reminded his hearers that it was not known in what degree the vomiting could be ascribed to nervous factors, histamine-like substances or other causes. He also spoke of the remarkable action of purely nervous factors on the stomach discovered by Pawlow and missed by Beaumont. In addition to vomiting, pain or even hæmorrhage might be produced by gastric neurosis. After quoting the Alvarez aphorism that there was a duplicate chemical plant in the alimentary canal but only one tube of transport, Professor Osborne emphasized the view that deficiency of pepsin was very rare and that though commercial preparations were on the market to supply a supposed deficiency, they were not of the slightest use whatever, and even if the patient had no pepsin the probability was that the added pepsin would be of no avail. On the other hand, a deficiency of hydrochloric acid was a serious matter and was capable of amelioration by appropriate treatment.

Professor Osborne then emphasized something which was not in the text-books about bile. Bile was non-toxic soap; water colour artists used ox bile to clear paper of grease. Beaumont had discovered the regurgitation into the stomach of concentrated bile, which proceeded to wash grease away from the food and let pepsin have a chance to act on the protein. It had to be remembered that bile, though non-toxic in the alimentary canal, was highly toxic elsewhere; a very little of it when spilled onto the peritoneum would produce all the effects of soap as an irritant and set up peritonitis. It had been observed constantly that when the duodenum or jejunum was opened froth or foam was found within. Remarkable physical processes were at work wherever foam was produced. It was the foam and not the agitation that produced butter in the process of butter making. The smear on the glass that had contained a mixture of milk and soda water was greater than that produced by milk alone; careful examination would reveal that the smear of milk and soda water consisted of bubbles of carbon dioxide in intimate contact with numerous satellites of butter. The foam found in the duodenum and jejunum had a big physical significance in connexion with the procedures happening in that section of the alimentary canal.

A fact of practical importance in the feeding of children was the next subject discussed by Professor Osborne. He said that the relative undevelopment of the pancreas was the secret of many of the digestive troubles in childhood. The casein of milk was the one protein that was digested in the absence of trypsin; it could be broken down by erepsin alone. Lactose did not require the pancreas, and the fat in milk could also be digested in conditions of loss or absence of pancreatic juices. The practical fact emerged that in the absence of lipase or steapsin the fat of milk could be digested, though that was not the case with any other form of fat. It was a common observation that a child might cut the fat off mutton or bacon, but would take liberal quantities of butter or cream. Patients after operations on the gall-bladder might boggle at ordinary fats or oils, but they could take butter or cream, which could be digested by the *succus entericus*.

Finally Professor Osborne directed attention to another point that to his knowledge had not been stressed anywhere. One and the same capillary in a gland or muscle had to bring the correct amount of oxygen to each cell and

to take away the correct amount of carbon dioxide. An excessive concentration of oxygen would kill even a cat, but if the amount of oxygen was inadequate the evils of anoxæmia became manifest. If carbon dioxide was removed too quickly, alkalosis would develop, with profound changes in the permeability of the cell membranes. Excess of carbon dioxide produced narcosis. Yet the one capillary had to do the double task. One little trick had been discovered by observation of the capillaries in the capillary network of frogs which was known as the "skimming action"; nobody had looked for it in the human being. That action could be carried out only if there was a fixed relation of the oxygen wanted to the carbon dioxide produced, and that meant a fixed relation of the fat consumed to the carbohydrate consumed. The presence of the carbohydrate in the capillary blood was well known, but it was not so generally recognized that fat was carried as ultra-microscopic globules. In the working cell the balance between the carbohydrate and fat utilized was that which just kept the proper balance between the oxygen required and the carbon dioxide produced. A. B. Macallum, of Toronto, had first drawn attention to the fact that inadequate combustion of organic material always resulted in the formation of tar derivatives as by-products, and those substances might be very clogging. A certain carburettor mixture must be supplied supplied to the cell. Irving Fisher had said that by instinct, not knowing what we did, we maintained the balance in the food. Professor Osborne was prepared to accept that dictum, though he was in doubt as to whether it applied in the case of the rice diet of the Chinese. Instinctively the intake of food was adjusted with nicety so far as the relative distribution of Calories derived from protein, carbohydate and fat respectively was concerned.

At the conclusion of his address Professor Osborne paid a tribute to his successor in the chair of physiology at the University of Melbourne, Dr. R. D. Wright. He also mentioned that Dr. Wright was engaged in research work in the field which had been the subject of his address that night, and he asked that any influence from his colleagues in the medical profession should be used where the opportunity occurred to give the incoming professor every opportunity of access to clinical material, so that he could become the unique professor of physiology, who could apply physiology to clinical problems and *vice versa*.

Professor Osborne's last statement was that the furred tongue remained a perpetual humiliation to the members of the medical profession.

Obituary.

JOHN GORDON.

WE are indebted to Dr. H. Boyd Graham for the following account of the career of the late Dr. John Gordon.

A life of fine endeavour and achievement ended with the death of the Melbourne surgeon, John Gordon, which was announced recently. He was the son of Mr. John Gordon, building contractor, and his early years were spent in Carlton, where he was born in 1870, and at King's College, East Melbourne, where his education was commenced. He entered on his medical course at the University of Melbourne in 1887 and graduated with final honours Bachelor of Medicine in 1891 and Bachelor of Surgery in 1892. During the years between he showed many evidences of great ability, gaining first-class honours in anatomy and winning the exhibition in pathology. Placed second in his year, he was prominent as one of the twelve resident medical officers at the Melbourne Hospital after graduation and gained further experience by visiting England, where he succeeded in obtaining the diplomas of Licentiate of the Royal College of Physicians and Member of the Royal College of Surgeons of England in 1894; in 1895 he gained the Fellowship of the Royal College of Surgeons of England. He then returned to his native city and commenced private practice in Carlton. In 1899 he gained the M.D. degree and in 1903 the M.S. degree of the University of Melbourne. He also served his hospital for many years as surgeon to out-patients and in 1911 as surgeon to in-patients, in which capacity he was busily engaged until he answered the call to war service. He had built up a great reputation as a clinical instructor of medical students and as a competent master of the surgical art.

John Gordon volunteered promptly for service with the Australian Expeditionary Force in 1914, and in March, 1915, was posted to the First Australian Clearing Hospital, under Lieutenant-Colonel W. W. Giblin, which became the casualty clearing station on the beach at the landing on Gallipoli. Arising out of the exigencies of service in Egypt and in the Dardanelles theatre of war Gordon slowly but surely developed evidences of rheumatoid arthritis and chronic bronchial asthma, which were to cloud his future. He was no longer a young man, and the living conditions were extremely arduous, apart altogether from the great pressure of surgical work to which he devoted himself wholeheartedly. Later came active service further abroad, in France and England, with the decoration order of a Campanion of the Most Distinguished Order of Saint Michael and Saint George awarded to him in 1918 for distinguished services, which were mentioned in official dispatches.

The War over, Gordon returned to resume practice as a surgeon in Collins Street and to his old position at the Melbourne Hospital. In spite of disability and pain he made himself carry on and did not complain. His store of wisdom was lavished on resident surgeons and medical students, much to their lasting benefit, and he made a passion of his very valuable work for the Limbless Soldiers' Association. His friends were delighted to hear of his appointment as surgeon to the Railways Department in 1924, when he resigned from his duties at the Melbourne Hospital. The appointment was most appropriate because of the outstanding qualities he was known to possess for fearless expression of surgical opinions and judgement, and unbounded patience and sympathetic understanding of the human element involved in his work. Unable to do any operative surgery because of the progressive crippling of his joints, Gordon raised the railways position to a very high level and maintained it until he had to retire in 1934.

John Gordon had two ambitions: to be an in-patient surgeon at the Royal Melbourne Hospital, and to be President of the Victorian Branch of the British Medical Association. We have seen that he achieved the former, and soon after his return from the War his fellows made him their President. In his address as the outgoing President in December, 1922, he spoke of the trends of medical practice and laid particular emphasis on the encroachment of public hospital and contract practice on private practice; half the population was treated either by contract practice or by charity, despite the apparent prosperity of the country.

The all-round soundness of John Gordon may be gathered from the additional information that during his career at the university he was very popular and was a fine sportsman. On two occasions he led the cricket eleven to victory against the University of Sydney, and for several years he was a member of the lacrosse team of his university. In May, 1907, he married Miss Ada Waters, of Brisbane, and found in her a splendid helpmate to support him in his heroic struggles to surmount fortune's deadly blows and to share with him the true dignity of his tranquil mind. She is still with us, he has passed on; in honouring his memory we shall help her in her bereavement.

Dr. R. C. Brown writes:

By the death of John Gordon on January 18 the medical profession has lost a quite exceptional personality and an altogether stirling character. To me, who have known him well for fifty years, he has been a hero.

We were students together in the late eighties and early nineties, when Jack Gordon was always a popular leader. He was a fine cricketer and lacrosse player, and was usually either first or second in the honours lists, excelling especially in anatomy and in pathology.

When he began private practice in Carlton they were hard times in Melbourne, but with Scottish dourness he chose probably the most difficult spot in which to begin life. He went on to gain the senior medical and surgical degrees and honorary appointments at the Melbourne Hospital while hard at work in Carlton, and later practised in Collins Street.

Though forty-five years of age, he left Melbourne with the first contingent of the Australian Imperial Force, and poor Gordon's real troubles began. I should here like to say that, though I was a close personal friend, I have never heard him utter one word of complaint. He contracted a tropical infection in Egypt, but landed at Gallipoli on April 25, 1915, and with characteristic determination, though much troubled with a weak heart, he carried on to the evacuation. In England a severe asthma began, which lasted a number of years, and when in France an osteoarthritis became evident, which gradually affected all his joints and forced his resignation from the Melbourne Hospital in 1924, owing to hand deformity.

For a number of years he acted as surgeon to the Limbless Soldiers' Association and retained this position until shortly before he died. The limbless soldiers were pets of his; ill as he was, he would attend them and their wives and families, and secured for them all kinds of special benefits.

In May, 1907, he married Miss Ada Waters, who proved a loyal and devoted and cheerful wife through the long years of suffering.

John Gordon was a fine, practical, highly qualified surgeon and, had Fate been kinder to him, would doubtless have occupied the highest surgical position for which he had prepared himself so well. But I wish to refer more to his personal qualities. I never heard him mention the disappointment he must have felt at leaving the hospital scene; the pain of his deformed joints and the distress of his asthma he bore without a complaint; he was always the same cheerful friendly companion, and even at the very last a little humour brought forth his ready smile. He was a generous friend and brother, who always seemed to disdain material advantages for himself. He was a man to whom his friend could go secure in the knowledge that Jack Gordon would disregard his own affairs and give his undivided attention to the other's troubles; and his advice was not that of an academist, but of an experienced man of the world.

Honest in his mind and in all his dealings, there was about John Gordon a complete absence of all humbug and pretence. It was characteristic of him that he died as he lived, thinking of the welfare of others, and he specially directed that there should be no funeral pomp and no mention of distinctions or honours in the death notice—just plain Dr. John Gordon.

Dr. Hume Turnbull writes:

Dr. John Gordon was appointed surgeon to in-patients at the Melbourne Hospital just before the War, to which he went with the First Stationary Hospital, so that the Melbourne Hospital was deprived of his services till 1919, and shortly afterwards he began to suffer the crippling effects of the arthritis of the hands which caused him to abandon surgery and resign his hospital appointment in 1924. This was a great loss to the hospital, not only because it was deprived of his wise opinion and skilful surgery, but because his personal influence was exerted always for gracious unselfish relations amongst the members of the staff, while the loss to the clinical school was a severe one.

His disability prevented him from doing much operating, and most of this had to be done by the junior surgeon of the team; but he was tireless in helping his house surgeons and assisting them with minor operations, while as a teacher of students he was excellent, kind, painstaking and very wise.

To all his colleagues, both senior and junior, he was courteous, generous and absolutely unselfish, and it can be said with confidence that he was loved by all and that his early resignation was regretted deeply.

While on service he was just the same, and everyone who was fortunate to be associated with him in any way realized his worth. He took an intense interest in the welfare of the limbless soldiers and made a special study of their problems, and on his return to Australia he worked very hard on the problem of amputation stumps and artificial limbs, and was in charge of much of the work at the factory for artificial limbs in Melbourne. Here and later, while he was railways medical officer, his kindness, courage and fairness endeared him to all the men under his care.

John Gordon was a fine character in every way, wise, learned and energetic, scrupulously fair and honest in all his dealings, and it was impossible to imagine his doing anything which was not absolutely straight or saying a

mean or unkind thing about anyone. His patience throughout his crippling and painful illness and the pluck with which he carried on until he felt that he could no longer do justice to his job was wonderful.

Dr. R. H. Fetherston writes:

In August, 1914, when war was declared, Gordon was one of the younger surgeons on the staff of the Melbourne Hospital. He volunteered at once. Because of his surgical knowledge, his general health and active disposition, he was attached as surgeon to the First Casualty Clearing Station, and sailed in 1914 in the *Kyarra*. Landing with the unit directly after "the landing" on Gallipoli, he remained there until the evacuation. At times the casualty clearing station practically became a hospital, the only Australian unit of its kind on Gallipoli. Gordon did fine work, but the stress and strain, together with infected food, told its tale. Like many others, he received a vital blow, not from shot and shell, though always under fire, but from a rheumatoid infection, which crippled and finally killed him, one of the forms of rheumatic infection which affected his heart and muscles, later attacking his hands and joints. When I saw him on Anzac, towards the end of 1915, his heart was giving trouble, and he could only get about slowly. I saw he was unfit and wanted him to return with me, offering to find him a suitable position where he could be properly cared for; but "no", he begged me not to mention his condition. He wanted to see either the final advance or the evacuation then being talked of.

By the time he got away and got to London—even London was no place for him—he was worse, but would not give up. He was posted to a hospital for crippled men and men being fitted with artificial limbs. It was a difficult unit to handle; but he was one of the medical officers for whom the men would do as they were told.

Gordon took up work on return to Australia, both in practice and at the Melbourne Hospital, but was not fit for either. His deformed hands soon forced his resignation from full indoor surgeon to the hospital. The disease progressed until he was finally bedridden and unable to help himself. He never complained, making the best of everything, not wanting anything said about his work or his suffering. He wanted no one to fuss about him, refused to have nurses, being satisfied that his wife could do everything for him that he wanted, and do it better than anyone else. He was probably right, for few permanently incapacitated men are so fortunate as to receive the devoted care and ceaseless attention that Gordon had bestowed upon him.

Post-Graduate Work.

GENERAL REVISION COURSE AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that the annual general revision course will be held in Sydney from Monday, March 27, to Thursday, April 6, 1939. The programme is set out hereunder.

Monday, March 27.

At the Robert H. Todd Assembly Hall, 135, Macquarie Street: 9 to 10.15 a.m., registration *et cetera*; 10.15 to 10.45 a.m., "Medico-Legal Problems", Dr. Clarence Read; 11.15 a.m. to 12 noon, "Anæsthesia and Analgesia in Labour", Dame Constance D'Arcy; 12 noon to 12.45 p.m., "Tonics and Sedatives", Dr. Harold Ritchie.

At Sydney Hospital: 2.15 to 3.15 p.m., "The Treatment of Pneumonia", Dr. A. Holmes à Court; 3.30 to 5 p.m., "Urological Emergencies", Dr. Reginald Bridge.

Tuesday, March 28.

At the Women's Hospital, Crown Street: 9.30 to 10.15 a.m., "The Toxæmias of Pregnancy", Dr. A. J. Gibson; 10.15 to 10.45 a.m., "Delayed Labour", Dr. R. D. Bowman; 11 to 11.45 a.m., "Ante-Partum Hæmorrhage", Dr. T. Dixon Hughes; 11.45 a.m. to 12.15 p.m., "Puerperal Sepsis", Dr. J. Chesterman; 12.15 to 12.45 p.m., "Some Applications of Clinical Pathology", Dr. Beatrix Durie.

Wednesday, March 29.

At the Royal North Shore Hospital: 9.30 to 10.30 a.m., "The Management of Diabetes", Dr. W. Wilson Ingram; 10.45 to 11.45 a.m., "The Early Recognition of Melancholia", Dr. D. W. H. Arnott; 11.45 a.m. to 12.45 p.m., "The Angina of Effort", Dr. A. J. Hood Stobo.

At the Royal North Shore Hospital: 2 to 3 p.m., "The Diagnosis of Spinal Cord Lesions", Dr. R. A. Money; 3 to 4 p.m., demonstration of cases illustrating chest conditions other than tuberculosis, Dr. G. Bruce White and Dr. Douglas Anderson.

At the Robert H. Todd Assembly Hall, 135, Macquarie Street: 8 p.m., cinematograph films.

Thursday, March 30.

At Saint Vincent's Hospital: 9.30 to 10.30 a.m., "Modern Aspects of Surgery of the Chest", Dr. V. M. Coppleson; 11 to 11.30 a.m., "The Use of Vitamins in General Medicine", Dr. R. Jeremy; 11.30 a.m. to 12.15 p.m., "The Management of Intestinal Obstruction", Dr. W. Maxwell; 2.15 to 12.45 p.m., "Anatomy in Minor Surgery".

At Saint Vincent's Hospital: 2.15 to 3 p.m., demonstration of the use of the gastroscope, with special reference to gastritis, Dr. Sherwood; 3.15 to 4.15 p.m., "Blood Transfusions and Methods of Typing", Dr. A. H. Tebbutt.

Friday, March 31.

At the Royal Alexandra Hospital for Children: 9 to 10 a.m., removal of tonsils, Dr. A. W. Dunn, Dr. Ramsay Beavis, Dr. C. Wesley; 10.15 to 11.15 a.m., "Whooping Cough", Dr. Edgar Stephen; 11.30 a.m. to 12.30 p.m., selected surgical cases, Dr. T. Y. Nelson.

At the Royal Alexandra Hospital for Children: 2.15 to 3 p.m., demonstration of skin tests used in clinical medicine, Dr. Phyllis Anderson; 3.15 to 4 p.m., "Injuries around the Elbow Joint", Dr. R. L. Stephen; 4 to 4.45 p.m., selected medical cases, Dr. Laurence Hughes.

Saturday, April 1.

At Sydney Hospital: 9.30 to 10.30 a.m., "Fractures", Dr. G. Bell and Dr. W. E. Kay; 10.30 to 11.30 a.m., "Fractures", Dr. A. M. McIntosh and Dr. C. E. Winston.

Monday, April 3.

At Lewisham Hospital: 9.30 to 10.30 a.m., "Hæmatemesis and its Treatment", Dr. C. G. McDonald; 10.30 to 11.15 a.m., "Renal Pain", Dr. Richard Flynn; 11.30 a.m. to 12.30 p.m., "Practical Aspects of the Neuroses", Dr. J. A. H. McGeorge.

At Lewisham Hospital: 2.15 to 3.15 p.m., "Ocular Emergencies", Dr. E. A. Brearley; 3.15 to 4.15 p.m., "Acute Appendicitis", Dr. T. W. Lipscomb.

Tuesday, April 4.

At the Prince Henry Hospital: 9.30 to 10.30 a.m., "Coronary Disease and its Treatment", Sir Charles Blackburn; 10.30 to 11.15 a.m., "Some Recent Methods in Anæsthesia", Dr. H. J. Daly; 11.30 a.m. to 12.30 p.m., selected medical cases, Dr. S. A. Smith.

At the Prince Henry Hospital: 2 to 3 p.m., "Infectious Diseases", Professor H. K. Ward; 3 to 4 p.m., "Some Surgical Aspects of Jaundice", Dr. H. R. G. Poate.

Wednesday, April 5.

At the Royal Prince Alfred Hospital: 9.30 to 10.30 a.m., "Drugs Used in Cardiac Disease: Their Use and Abuse", with illustrative cases, Dr. A. J. Collins; 10.45 to 11.45 a.m., "Acute Inflammation of the Meninges", Dr. Allan Walker; 11.45 a.m. to 12.45 p.m., "Recognition and Management of Acute and Chronic Diseases of the Ear", Dr. R. S. Godsall.

At the Royal Prince Alfred Hospital: 2.15 to 3.30 p.m., dermatological demonstration, Dr. E. H. Molesworth; 3.45 to 4.45 p.m., demonstration of cases and results of treatment of pulmonary tuberculosis, the Anti-Tuberculosis Staff (Dr. W. A. Bye, Dr. W. P. MacCallum, Dr. N. H. W. Saxby and Dr. T. Rofe).

Thursday, April 6.

At the Royal Hospital for Women: 9.30 to 10.15 a.m., "The Care and Feeding of Infants", Dr. Margaret Harper; 10.15 to 10.45 a.m., "Late Post-Partum Hæmorrhage", Dr. P. L. Hipsley; 11 to 11.45 a.m., "Some Recent Advances in Obstetrics", Professor J. C. Windeyer; 11.45 a.m. to 12.30 p.m., "Trichomonas Vaginalis", Dr. F. Brown Craig.

The fees for the course will be as follows: full course of two weeks, three guineas; mornings or afternoons only, two guineas; one week only, two guineas. For hospital resident medical officers and those not engaged in actual practice the fees for the course will be one-half of those shown above.

Application, which should be accompanied by a cheque for the amount of the fee, should be made to the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay.

Correspondence.

THE ALLEGED PAINFUL SEQUELÆ AND POOR RESULTS OF ANTROSTOMY.

SIR: I read Dr. A. B. K. Watkins's excellent article of February 25, 1939, with great interest, and would suggest that all medical practitioners might read it with profit and reconsider the absurdly prevalent prejudice against any antrum operation.

I agree with Dr. Watkins in almost every respect, but would like to add one or two remarks.

In my opinion a lot of the post-operative swelling is due to packing the antrum after a radical operation; I know that since I have ceased to pack either the nose or antrum I have had negligible swelling as an after-result.

I would like to urge every practitioner to look for antrum infection in its early stages, because if they do recognize it there will be very little antrum surgery necessary.

Yours, etc.,
R. H. BETTINGTON.

185, Macquarie Street,
Sydney,
February 28, 1939.

YAWS AND SYPHILIS AMONG AUSTRALIAN ABORIGINES.

SIR: There is still much confusion about the relative incidence of the diseases yaws and syphilis amongst the Australian aborigines. The following figures and comments are rendered in the hope that they may lead to a more comprehensive survey and study of the problem. Wassermann and Kline tests were performed on one hundred and twenty-five natives of the Kimberley district of Western Australia.

While we did recognize yaws as a clinical entity, fairly common amongst the children, who showed typical sores around the mouth, natal cleft and axillæ, we found syphilis often difficult to diagnose clinically, due to the fact, amongst others, that the great prevalence of *granuloma pudendi*, with its widespread ulceration and resultant scarring, often masked the picture. However, syphilis was unmistakable in twenty-five out of seventy-nine cases giving positive Wassermann and Kline reactions. Tabulated as follows:

Wyndham District.		*Broome District.*	
A. Primary chancre	1	*A.* Primary chancres	4
B. Tertiary lesions—		*B.* Tertiary lesions—	
a. Tabes dorsalis	1	*a.* Perforated palates	2
b. Ulcerating gummata of legs *et cetera*	3	*b.* Primary optic atrophy	1
c. Perforated palate	1	*c. Tabes dorsalis* with perforating ulcer of foot	1
C. Contacts of whites then with proven syphilis	4		
D. Contacts of Group *A*	3		
E. Contacts of Group *C*	4		
Total	17	Total	8
Total number bloods Wyndham series equals 24, and all these gave positive Wassermann and Kline reactions.		Total number in Broome series 101, of which 55 were positive.	

Twenty-five of these cases can be considered suffering from syphilis in a primary or tertiary form. Of the remaining 54 cases some have been under treatment, and blood tests have remained positive in six cases after 4·8 grammes of "Novarsenobillon", 16·0 cubic centimetres of "Muthanol" had been given. These are the only retests we have been able to do up to date, and it is probable that these people are also syphilitics.

In conclusion we might say that we consider that the incidence of syphilis is high enough to assume practical importance amongst the natives. It is probable that the fall in birth rate amongst the Kimberley tribes is due in some extent to syphilis, although gonorrhœa and granuloma must play a part.

The spread of venereal disease in general in the Kimberleys is due to the type of low-grade white man (commonly known as dead-beat) that is allowed to associate with the natives.

Further work is being carried on through the Commonwealth Health Laboratory functioning at Broome, and we would welcome any discussion and advice on the subject.

Yours, etc.,
A. J. KING, B.Sc., M.B., B.S.,
District Medical Officer, Wyndham, 1936-1938.
F. K. WALLACE, M.B., B.S.,
District Medical Officer, Broome.

Undated.

Congresses.

INTERNATIONAL CONGRESS OF HYDROLOGY, CLIMATOLOGY AND MEDICAL GEOLOGY.

THE sixteenth International Congress of Hydrology, Climatology and Medical Geology will be held at Strasburg on October 8, 9, 10 and 11, 1939. The Presidents will be Professor Danjou, of the Faculty of Science; Professor Sartory, of the Faculty of Pharmacy; and Professor Forster, of the Faculty of Medicine. Among the subjects to be discussed at the congress are: "Permeability and Mineral Waters", "The Hydromineral Treatment of the Dermatoses", "The Indications and Contraindications for Winter Sports", "Mineral Waters of Petrol-Yielding Districts". The General Secretary will be Professor Vaucher, at Institut d'Hydrologie et de Climatologie, 1 place de l'Hôpital, Strasburg. Further information may be obtained from Professor Vaucher.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Gulson, Gordon Leslie, M.B., B.S., 1939 (Univ. Sydney), Tamworth Hospital, Tamworth.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Burniston, George Garrett, M.B., B.S., 1939 (Univ. Sydney), Hornsby and District Hospital, Hornsby.
Campbell, Neil Douglas, M.B., B.S., 1939 (Univ. Sydney), St. George District Hospital, Kogarah.
Davidson, Charles Geoffrey, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.
Davis, Eric Lewis, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.
Edwards, Margaret Jean, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.
Finckh, Dorrie Alfreda, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.
Hipsley, Eben Hamilton, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Isbister, James, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Moyes, James Murray, M.B., B.S., 1939 (Univ. Sydney), St. George District Hospital, Kogarah.
Oldham, Jack Maxwell, M.B., B.S., 1939 (Univ. Sydney), Goulburn District Hospital, Goulburn.
Paton, Jean Sinclair, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Prior, Herbert James, M.B., B.S., 1939 (Univ. Sydney), Sydney Hospital, Sydney.
Vickery, Ian Firth, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Watson, Douglas Godfrey, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned has applied for election as a member of the Tasmanian Branch of the British Medical Association:

Cunningham, Anthony Benedict, M.B., Ch.M., 1921 (Univ. Sydney), 119, Macquarie Street, Hobart.

Diary for the Month.

MAR. 28.—New South Wales Branch, B.M.A.: Council (Quarterly).
MAR. 30.—South Australian Branch, B.M.A.: Branch.
MAR. 30.—New South Wales Branch, B.M.A.: Annual Meeting.
APR. 4.—New South Wales Branch, B.M.A.: Council.
APR. 5.—Western Australian Branch, B.M.A.: Council.
APR. 5.—Victorian Branch, B.M.A.: Branch.
APR. 6.—South Australian Branch, B.M.A.: Council.
APR. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 14.—Queensland Branch, B.M.A.: Council.
APR. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee and Ethics Committee.
APR. 19.—Western Australian Branch, B.M.A.: Branch.
APR. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
APR. 26.—Victorian Branch, B.M.A.: Council.
APR. 27.—New South Wales Branch, B.M.A.: Branch.
APR. 27.—South Australian Branch, B.M.A.: Branch.
APR. 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi to xviii.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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